VOL. VIII. No. 4

BRITISH HEART JOURNAL

EDITORS
MAURICE CAMPBELL
D. EVAN BEDFORD

APPOINTED BY
THE BRITISH CARDIAC SOCIETY

CONTENTS

Anæmi Visnagi in the Treatment of Angina Pectoris. By G. V. Anrep, G. S. Barsoum, M. F. Kenady and G. Mikaberidze . . . 171

Premature Systoles originating in the A-V Node. By R. J. Lascelles and S. S. Mehta . . . 176

Dissecting Aneurysm with Spontaneous Rupture of Coronary Infarction. By Hyslop Bell and P. L. Mills . . . 181

Chronic Dissecting Aneurysm. By A. Morgan Jones and F. A. Langley . . . 194

Dissecting Aneurysm with Survival after Rupture into Pleural Cavity. By Maurice Campbell . . . 200

Dissecting Aneurysm of the Ascending Aorta . . . 203

Aneurysmal Dilatation of the Left Auricle and Bronchial Spine. By H. Ashworth and A. Morgan . . . 207

Early Diagnosis of Rheumatic Valvular Disease. By John Parkinson and Ronald Hartley . . . 212

Proceedings of the British Cardiac Society . . . 233

Index . . . 239

LONDON
BRITISH MEDICAL ASSOCIATION
TAVISTOCK SQUARE, W.C.1

PUBLISHED QUARTERLY

AMMI VISNAGA IN THE TREATMENT OF THE ANGINAL SYNDROME*

BY

G. V. ANREP, G. S. BARSOUM, M. R. KENAWY, AND G. MISRAHY

From the Physiological Laboratory and the University Hospital, Cairo, Egypt

Received August 28, 1945

The plant *Ammi Visnaga*, known in Arabic as " Khella," grows wild in the Eastern Mediterranean countries and in Arabia. The decoction of its dried seeds is much used by the population and is frequently prescribed by local physicians as a diuretic and antispasmodic in cases of ureteral stones. Of the several crystalline substances isolated from the seeds, quantitatively the most important are a substance called khellin ($C_{14}H_{12}O_5$) and a monoglucoside ($C_{19}H_{20}O_5 2H_2O$). Almost the entire activity of *Ammi Visnaga* is due to the khellin.

Khellin was first prepared in an impure form by Mustapha (1879) and again by Malosse (1881). Fantl and Salim (1930) isolated and purified both khellin and the glucoside; they determined their respective properties and composition, established methods for their separation, and suggested the most likely structural formulæ. The pharmacognosical features of the two substances were studied by Fahmy (1931). The final determination of their structural formulæ was made by Malik (1932) for khellin and by Hassan (1932) for the glucoside, both working under the direction of Prof. R. W. West of Cairo University. In 1934 the decoction and the tincture of *Ammi Visnaga* were included in the Egyptian Pharmacopœia. Pharmacologically the action of khellin was studied by Samaan (1932) who showed that it relaxes all the visceral smooth muscles by direct action on the muscle fibres. Samaan also suggested changing the name of khellin to visamin. We prefer the original name since it is free from the implication of containing an amino group. Preliminary experiments of Anrep and Misrahy (1945) showed that khellin is a strong coronary vasodilator.

The object of the present research was to investigate by physiological methods the action of khellin on the cardiovascular system with special reference to the coronary circulation and the heart muscle, and also to study its action clinically on patients suffering from angina pectoris and coronary thrombosis.

I. Physiological Part

The experiments were made on dogs, using the heart–lung preparation and the whole animal. The coronary blood was collected from the coronary sinus through a Morawitz cannula and the blood flow was registered by means of a small volume recorder. The khellin at our disposal was a crystalline product of a constant melting-point (153° C.). We should like to thank Prof. Samaan for the supply of a small sample of the active material for our preliminary experiments. The rest of the experiments and almost all the clinical observations were made with khellin prepared in the Physiological Laboratory by a modified method of Fantl and Salim. The results of our observations are best presented by a description of a few typical experiments.

Experiments on the Heart–Lung Preparation

Experiment 1. Heart–lung preparation; blood pressure 100 mm. Hg.; cardiac output 500 c.c. per minute; temp. 36·5° C. The coronary sinus outflow was steady at 41–42 c.c. per minute. On administration of 10 mg. of khellin the coronary blood flow rapidly increased to

*A preliminary communication was published in the *Gazette of the Faculty of Medicine*, Cairo, 1945, 13, 39, by Kenawy and Barsoum.

120 c.c. per minute. The blood flow remained increased up to the end of the experiment, over 3 hours, fluctuating between 110 and 130 c.c. per minute. The total amount of blood in the circulation was about 1 litre and the weight of the heart 85 g. The record obtained in this experiment is reproduced in Fig. 1.

Experiment 2. Blood pressure 95 mm. Hg.; cardiac output 450 c.c. per minute; temp. 37° C. The coronary blood flow was steady at 33 c.c. per minute. After administration of 1 mg. of khellin dissolved in 5 c.c. of blood it increased to 50 c.c. per minute; after another 1 mg. it became 70 c.c. per minute. A further administration 10 minutes later of 3 mg. increased the flow to 100 c.c. per minute. The heart rate and the systemic output remained constant while the increase in the coronary circulation was maintained to the end of the experiment. The amount of blood in circulation was about 1 litre and the weight of the heart 74 g. A record of this experiment is given in Fig. 2.

Experiment 3. This experiment was made in order to determine the minimal active dose of

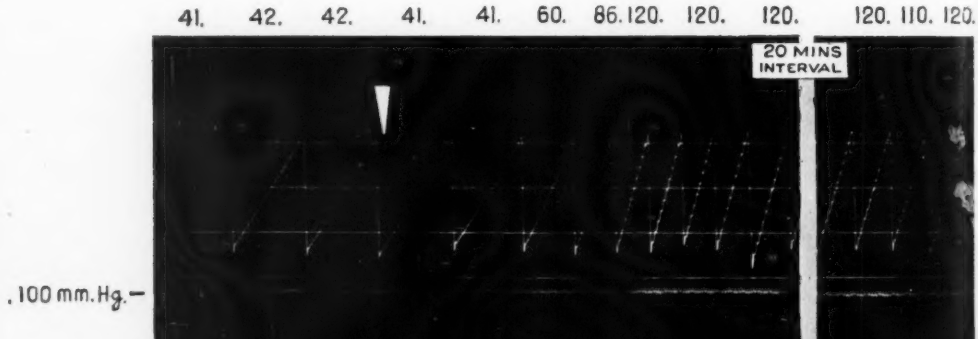


FIG. 1.—*Expt. 1.* Heart–lung preparation. Shows outflow from coronary sinus registered by volume recorder calibrated in 5 c.c. The figures added above the smoked paper give the flow in c.c. per minute. Blood pressure and time in seconds are recorded below. The arrow indicates injection of 10 mg. khellin.

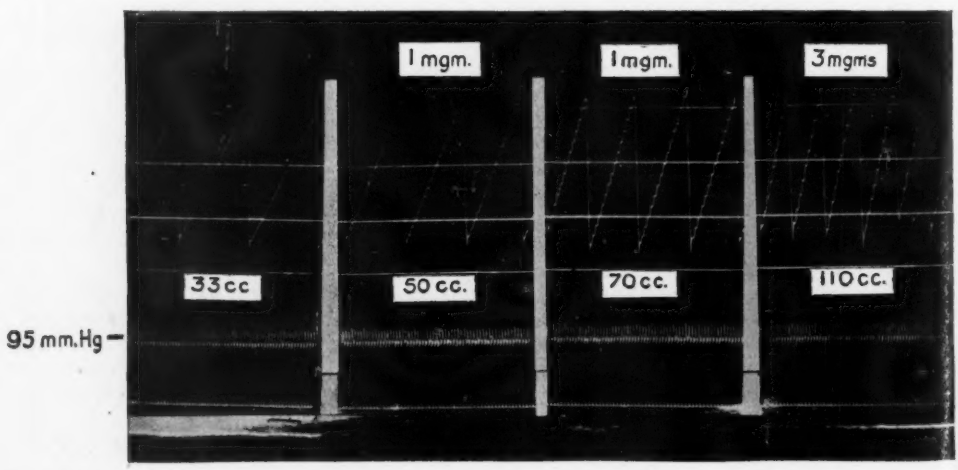


FIG. 2.—*Expt. 2.* Heart–lung preparation. Shows effect of small doses of khellin (upper figures) on coronary flow, registered by volume recorder and given in c.c. per minute in lower figures. The records were taken at intervals of 10 minutes.

khellin that would dilate the coronary blood vessels. The conditions of the experiment were like those of experiments 1 and 2. The amount of blood in circulation was measured as exactly as possible. The khellin was administered in successive small doses at intervals of 5 to 10 minutes.

Mg. of khellin administered	Approximate concentration	Coronary blood flow in c.c. per minute
None	None	44–46
0·4	1 in 2,500,000	47–49
0·4	1 in 1,250,000	56–57
0·8	1 in 625,000	83–85
3·2	1 in 208,000	120–130
3·2	1 in 125,000	160–180

It can be seen from the above and other similar experiments on the dog that the minimal active concentration of khellin in the heart–lung preparation is about 1 in 2,000,000. With concentrations of the order of 1 in 200,000 the coronary blood flow increases 3 to 4 times the initial volume. The action of khellin, although very considerable, is less than that of amyl nitrite, but it has the advantage of being much more prolonged. We should like to add that the isolated perfused rabbit's or cat's heart is not a suitable object for the study of the coronary dilator action of khellin. As with many other substances the results obtained are indefinite and inconclusive.

Samaan (1932) reports that in high concentrations khellin causes a weakening of the isolated perfused frog's and rabbit's heart. In repeated experiments on the heart–lung preparation no such effect was observed. Gradual administration of doses as high as 100 mg. causes no change in the heart volume, which is generally recognized as the best measure of cardiac fitness (Fig. 3). It is, therefore, obvious that doses many times greater than those causing a conspicuous coronary vasodilatation have no injurious effects on the heart muscle. Cardiograms taken before and after administration of khellin were found to be identical in all respects. The pulmonary blood pressure and the rate of the denervated heart were not affected.

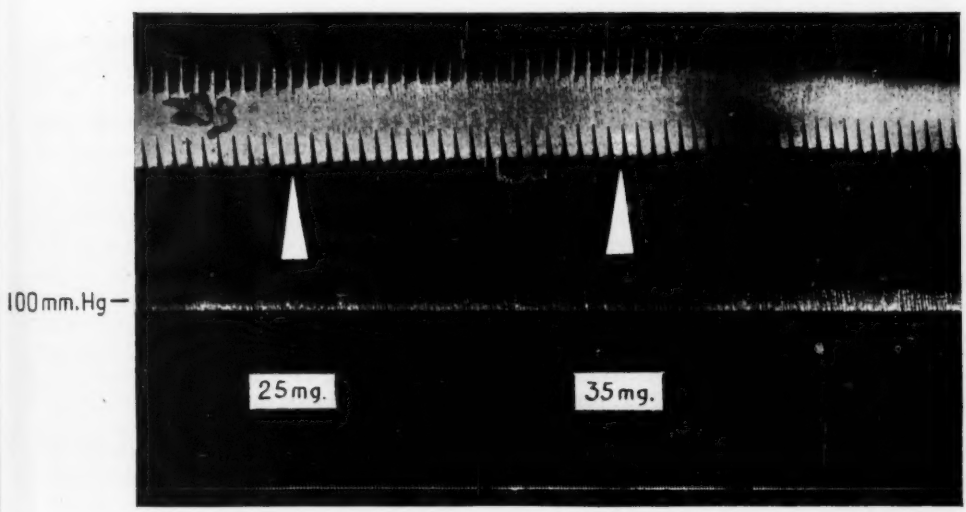


FIG. 3.—Heart–lung preparation. Effect of khellin on the heart volume; 25 and 35 mg. of khellin were administered at first and second arrow respectively.

Experiments on the Whole Animal

According to the observations of Samaan (1932) intravenous injections of khellin into the whole animal lead to a temporary fall of the arterial blood pressure, to a slowing of the heart rate which is vagal in origin, and to some acceleration of the respiration. Our experiments on the whole animal were made on dogs anæsthetized with chloralose (0·075–0·1 g. per kg.) or medinal (0·22 g. per kg.). The blood pressure was measured in the carotid artery while the respiration was recorded by a Marey's tambour which was placed on the sternum. Intravenous injections of khellin produced effects similar to those described by Samaan but only on rapid administration of large doses. Immediately after a rapid injection of 20–30 mg. the blood pressure drops to about 50 mm. Hg., the heart beats considerably slower, and the respiration is momentarily arrested. The entire effect lasts for only a short time; within a minute or two the general blood pressure, heart rate, and respiration return to normal. Slow intravenous injections at the rate of 2 mg. per 20–30 seconds can be continued for a long time without ill effects (Fig. 4). The maximal total dose so injected was 10 mg. per kilogram.

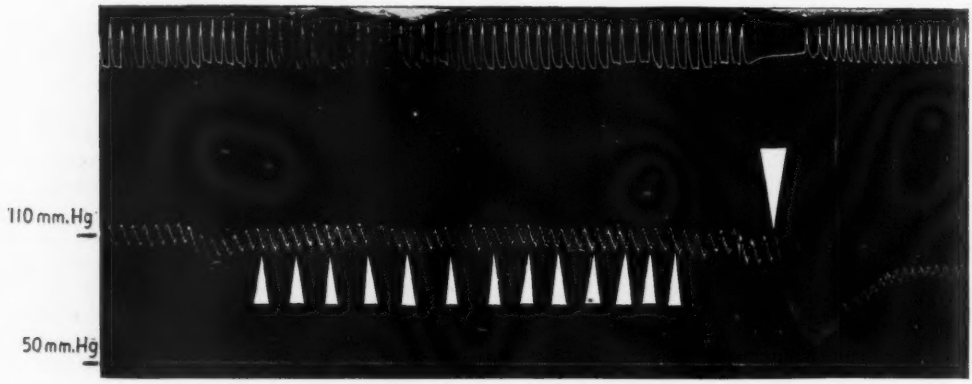


Fig. 4.—Effect of gradual and of rapid intravenous injections of khellin on the blood pressure and respiration of the whole animal. The end of injections of each 2 mg. is marked by an arrow pointing upwards. A rapid injection of 20 mg. was made at the last arrow pointing downwards. Time in seconds, arterial blood pressure, and respiration from below upwards.

It follows from these experiments that subcutaneous and intramuscular injections should be well tolerated. Doses of 40–50 mg. per kilogram cause a prolonged fall of the general blood pressure due to peripheral vasodilatation mainly of the splanchnic area.

The coronary vasodilator action of khellin can be observed not only in the heart–lung preparation but also in the whole animal. Experiment 4 is selected from amongst several to illustrate the effect.

Experiment 4. Dog, 11 kg., anæsthetized with chloralose and injected with chlorazol-fast-pink as anticoagulant. To prevent loss of blood the coronary cannula is connected with the jugular vein. It is opened to the volume recorder only during the measurements of the coronary blood flow. Before administration of khellin the coronary blood flow was steady at 27 c.c. per minute. After injection of 10 mg. the flow increased to 50 c.c. After injection of another 10 mg. the flow reached 80 and then 100 c.c. per minute. The general blood pressure remained unchanged at 105 mm. Hg. Towards the end of the experiment, one hour later, the coronary blood flow was 90 c.c. per minute and the arterial blood pressure, 95 mm. Hg.

Khellin exerts no stimulating effect on the sympathetic nervous system. It is, therefore, unlikely that its coronary vasodilator action is similar in nature to that produced by adrenaline. As with other smooth muscles it acts directly on the muscle fibres of the blood vessels. The apparently selective action of khellin on the coronary blood vessels is due to their much greater sensitivity to the drug as compared with the systemic blood vessels.

II. Clinical Part

As a clinical trial khellin was given to 46 patients of whom 38 had angina pectoris of effort or decubitus or both, and 8 had coronary thrombosis. The cases of angina pectoris were graded as mild, moderate, or severe. Those subject to occasional pain on effort were classed as mild; those with more frequent pain on effort and occasionally apart from it, who used trinitrine regularly, as moderate; those with frequent pain on slight effort or at rest and especially at night, who used trinitrine freely, as severe. On this basis, 8 cases were mild, 17 moderate, and 13 severe. There were 34 men and 4 women, and their ages ranged from 35 to 66 years. The duration of anginal symptoms was 3–12 years in 14 cases, 1–2 years in 17, and less than a year in 7. Hypertension was present in 15 cases and diabetes in 7. The Wassermann reaction was negative in all but one case. Abnormal electrocardiograms were recorded in 17 cases, and enlargement of the heart and aorta was found in 4 cases. Of the 8 cases of coronary thrombosis, 6 had recurrent attacks of pain, and 2 had dyspnœa during the period of complete rest following the onset of symptoms.

The subjective effects of the drug were recorded in each case. In some, electrocardiographic changes provoked by graded exercise were recorded before and after treatment. Exercise tolerance tests were performed whenever the patient's condition permitted. As a control, injections free from khellin or containing a much reduced dose were substituted without the patient's knowledge.

Method of Administration and Dosage. The following preparations were tried.

1. Purified liquid extract containing 40 mg. of active principle per c.c.; dose 30–60 minims in water with meals.
2. Pills each containing 25 mg.; dose 2–3 pills thrice daily after food.
3. Solution of 60 mg. per c.c. by intramuscular injection; dose 1–2 c.c. daily or on alternate days, and for the treatment of individual attacks.

Doses of 10–30 mg. per day by injection were tried first and found to be ineffective. A dose of 60 mg. a day gave encouraging results; and 90–120 mg. a day still better. Single doses of 150–200 mg. were given to healthy volunteers without ill effect. An average effective dose proved to be 90 mg. a day. The injections caused slight and transient local pain. The average oral dose was 40 mg. thrice daily.

Anginal Group

The response to khellin was arbitrarily classed as good, moderate, or negative; good when the anginal attacks ceased altogether or became very infrequent and mild, moderate when they diminished in frequency and severity, and negative otherwise. On this basis the results are summarized in Table I. The duration of observation and treatment varied from 3–9 months, being 6 months or longer in half, excluding those classed as failures where treatment was abandoned earlier.

TABLE I

Grade of Anginal Pain	Response to Treatment		
	Good	Moderate	Negative
Mild	7	1	—
Moderate ..	15	1	1
Severe	6	5	2
Total ..	28	7	3

Continuous Treatment. Oral administration of the liquid extract or pills alone was tried in 13 cases, intramuscular injections alone in 9, and both methods combined in 16. The 13 cases treated by oral doses of 120 mg. of khellin a day all showed a good response. Given

by mouth, the drug required 1–3 days to reach its full effect. The severity of the attacks was reduced before the frequency. If the drug was discontinued for a few days the attacks reappeared, sometimes in milder form.

Of 9 cases treated by injections alone, in doses of 90 mg. daily or 120 mg. on alternate days, 8 showed a good response. On cessation of the injections, or on substitution of inert injections as a control, the anginal attacks recurred, and restarting the injections again resulted in relief. The effect of each injection appeared to last 24–36 hours.

The 16 patients treated by a combination of oral and intramuscular khellin were mostly classed as severe cases. The injections and oral doses were given on alternate days. This combined treatment seemed more effective in severe cases, though two so treated failed to respond. One of these also had carcinoma of the stomach, and the other, after several days of frequent anginal attacks, died suddenly from coronary thrombosis. The third failure was treated orally for three weeks without effect.

Treatment of Individual Attacks. Patients were given liquid extract of khellin to take in the attacks, and nearly all reported favourably, especially in the case of more prolonged attacks of pain occurring apart from effort. In a few cases, we had the opportunity of giving injections during an attack and relief followed within a few minutes, rather more slowly than after trinitrine tablets.

Objective Tests. In some cases electrocardiograms before and after a standard exercise test of stepping on a chair 40 cm. high at a rate of 30 times a minute were recorded. Later,

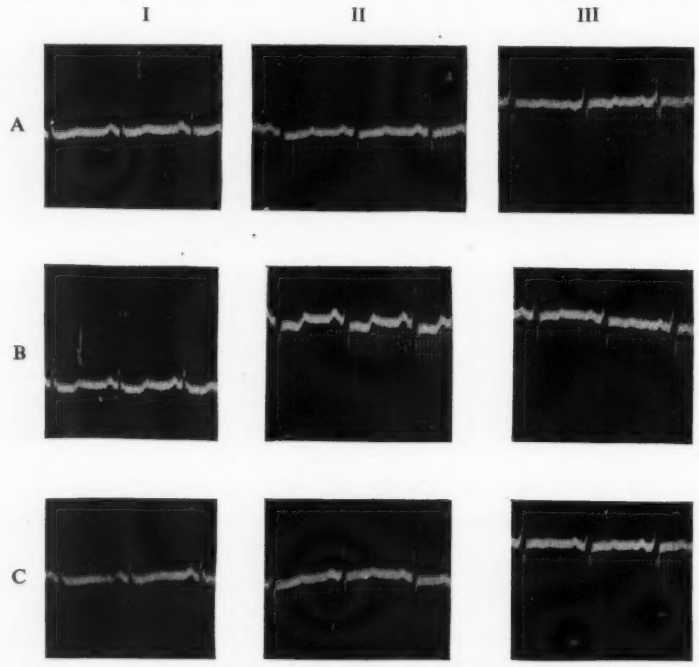


FIG. 5.—Effect of khellin on the electrocardiogram taken after standard exercise. (A) at rest; (B) after exercise; (C) taken on the following day, after a similar exercise, and 30 minutes after an intramuscular injection of 90 mg. of khellin.

the same exercise was performed 30 minutes after an injection of khellin, and the cardiogram was again recorded. Four cases showed S–T depression after a test exercise; in three of these, an injection of 90 mg. of khellin prevented the occurrence of S–T depression after the same exercise (Fig. 5), and in the fourth S–T depression was less conspicuous. In all patients tested, the exercise tolerance, as measured by the exercise described, increased after khellin.

Side Effects. A few patients complained of insomnia after the first few doses, one complained of pain and fullness in the head, and two of dyspepsia after oral administration. The blood pressure, pulse rate, and respiration were unaffected by doses up to 120 mg. of khellin. The clotting time and bleeding time were also unaffected.

Coronary Thrombosis Group

Khellin was given orally or by injection in doses of 90–120 mg. daily for 2–5 weeks after the onset of coronary thrombosis in 8 cases, in 6 of which there were recurrent anginal attacks during the period of recumbency. The drug was also given with morphine at the onset of symptoms. It was well tolerated and as far as could be judged it relieved the spasmodic anginal attacks that followed the initial attack of pain. The variability of the symptoms after coronary thrombosis makes it difficult to assess the effect of drug treatment, and the small number of cases yet treated is insufficient to warrant any definite conclusions as to the value of the treatment. It seems rational to give a coronary vasodilator that does not lower general blood pressure or affect the heart rate, with a view to preventing any associated coronary vasoconstriction, and thus minimizing the area of cardiac infarction.

Conclusions

Physiological experiments show that khellin is an effective coronary vasodilator in doses insufficient to cause any general fall in blood pressure. A preliminary clinical trial has shown that the drug may be given safely and without toxic effects in the doses prescribed. Khellin has advantages over the nitrites and other reputed vasodilators in that it has a selective action on the coronary vessels, and effective doses need not, therefore, lower systemic blood pressure. Its action is slower but more prolonged than that of the nitrites. It may be given in the form of continuous treatment to abolish or reduce the frequency and severity of anginal attacks, or to relieve individual severe attacks of pain. It is no simple matter to assess the value of a drug in the continuous treatment of angina pectoris, as Evans and Hoyle (1933) have shown, for they found that even a placebo might produce subjective improvement in a surprisingly large proportion of cases. This preliminary clinical trial, following physiological experiments, seems sufficiently favourable to justify a further and more extensive therapeutic test of khellin in angina pectoris and coronary occlusion.

Summary

Khellin, the active principle of *Ammi Visnaga*, has been tested physiologically in dogs, both on the heart–lung preparation and on the whole animal, in regard to its effect on the heart and coronary circulation. It was found to be an effective vasodilator with a selective action on the coronary vessels, so that coronary flow was increased by doses insufficient to lower the general blood pressure.

A clinical trial of khellin in 38 cases of angina pectoris and in 8 cases of coronary thrombosis is reported. Continuous treatment, by the oral or intramuscular routes or by both combined, gave favourable results in 35 out of 38 cases of angina pectoris. The drug was given continuously for several weeks to eight patients after coronary thrombosis and appeared to act favourably, but no definite conclusions could be reached as to its value in this respect.

References

Anrep, G. V., and Misrahy, G. (1945). *Gaz. Faculty Med.*, Cairo, **13,** 33.
Evans, W., and Hoyle, C. (1933). *Quart. J. Med.*, N.S., **2,** 311.
Fahmy, I. R., and Keiy, M. A. (1931). *Report of Pharmaceut. Soc. Egypt*, vol. 3.
Fantl, P., and Salim, S. I. (1930). *Biochem. Z.*, **226,** 166.
Hassan, M. K. (1932). Thesis, Cairo.
Malik, W. S. (1932). *Ibid.*
Malosse, T. (1881). Thèse, Montpellier.
Mustapha I. (1879). *C.R. Acad. Sci.*, Paris, **89,** 442.
Samaan, K. (1932). *Quart. J. Pharm. and Pharmacol.*, **5,** 6.

PREMATURE SYSTOLES ORIGINATING IN THE SINO-AURICULAR NODE

BY

R. LANGENDORF AND S. S. MINTZ

*From the Cardiovascular Department, Michael Reese Hospital, Chicago, Illinois, U.S.A.**

Received July 15, 1946

Premature systoles arising in the sino-auricular node are characterized in the electrocardiogram by premature P waves that are identical in contour with other sinus P waves and by the absence of a prolongation of the returning cycle. Actually the returning cycle may be shorter than the normal cycle because of prolonged sino-auricular conduction of the premature beat (Wenckebach, 1908). According to Wenckebach and Winterberg (1927) slight changes in contour of the premature P wave do not rule out sinus origin of the premature impulse; these aberrant P waves are ascribed to aberrant conduction in the auricles. Varying intra-auricular conduction has also been suggested (Scherf, 1945) to explain the altered contour of the sinus P wave that occurs in second degree auriculo-ventricular block, accompanied by shortening of the P–P intervals, in those P–P intervals containing a ventricular complex. It would appear that a contour of the premature P wave identical with that of the sinus P wave should be postulated for the diagnosis of sinus premature systoles in order to avoid confusion with auricular premature systoles. In exceptional cases, however, a diagnosis of sinus premature systoles seems justified in spite of a slightly different P wave contour when sinus arrhythmia is absent and when the returning cycle is equal to or shorter than the normal cycle. Because of their rarity, two more cases are being added in this paper to the few instances of sinus premature systoles reported (Wenckebach, 1908; Lewis, 1925; Clerc *et al.*, 1938; Simon and Langendorf, 1944; and Geiger and Goerner, 1945).

Case 1. A man, aged 60, was admitted to the hospital because of neurological complaints. A cardiogram (Fig. 1) was taken to determine the nature of his irregular heart action. The patient had received no medication. Lead I shows an undisturbed regular sinus rhythm with a P–P interval of 0·98 sec.; all the other leads show bigeminal rhythm with a P–P interval of 0·98 sec. alternating with a P–P interval of 0·80 sec. There is a first degree A-V block (P–R equals 0·30 sec.) with variations in the P–R (0·32 and 0·34 sec.) in the leads with bigeminal rhythm. The P waves show only minute differences in contour. The absence of a compensatory pause (the P–P interval in lead I equals the post-extrasystolic P–P intervals in the other leads) identifies the premature systoles as arising in the sino-auricular node. Comparison of lead I with the other leads permits exclusion of other possibilities to be considered in cases of persistent sinus bigeminy, such as non-conducted sinus premature systoles occurring after every second beat, 3 : 2 S-A block with the Wenckebach phenomenon, and alternans of the S-A conduction time.

Case 2. A woman, aged 80, having hypertensive cardiovascular disease, was admitted with a provisional diagnosis of a recent myocardial infarct. No drugs were administered. The cardiogram is shown in Fig. 2. The first seven beats in lead I are regularly spaced and have a P–P interval of 0·64 sec. They are followed by a bigeminal rhythm throughout the rest of the record except for one occasion in lead CF_2. A P–P interval of 0·64 sec. alternates with a P–P interval of 0·70 sec. However, the P wave of the premature beat shows minute differences in contour and might suggest ectopic rather than sinus origin. The first seven beats in lead I have to be considered as a run of premature systoles. This case illustrates the difficulty of distinguishing between sinus premature systoles and

* Aided by the Emil and Fanny Wedeles Fund for Cardiovascular Research. The department is supported in part by the Michael Reese Research Foundation.

FIG. 1.—Premature systoles originating in the S-A node.

auricular premature systoles arising close to the sino-auricular node. The diagnosis is based in this case upon the close similarity of the premature P waves to the sinus P waves and is made despite the inability to compare the post-extrasystolic pauses with the sinus P–P interval.

SUMMARY

The criteria of electrocardiographic diagnosis of premature systoles arising in the sino-auricular node are discussed and two new cases are described.

The diagnosis is made in spite of slight variations in the contour between the sinus P wave and the premature P wave. In Case 1 the diagnosis is based on the finding of a returning cycle equal to the interval between two sinus P waves in another lead without premature beats. In Case 2 the occurrence of bigeminal rhythm speaks against the diagnosis of simple sinus arrhythmia and favours the diagnosis of premature beats of sino-auricular origin because of the close similarity of the premature P waves to the sinus P waves; however, the diagnosis of auricular premature systoles arising from a focus close to the sinus node cannot be ruled out.

We are indebted to Dr. L. N. Katz for his criticism and aid in preparing this paper.

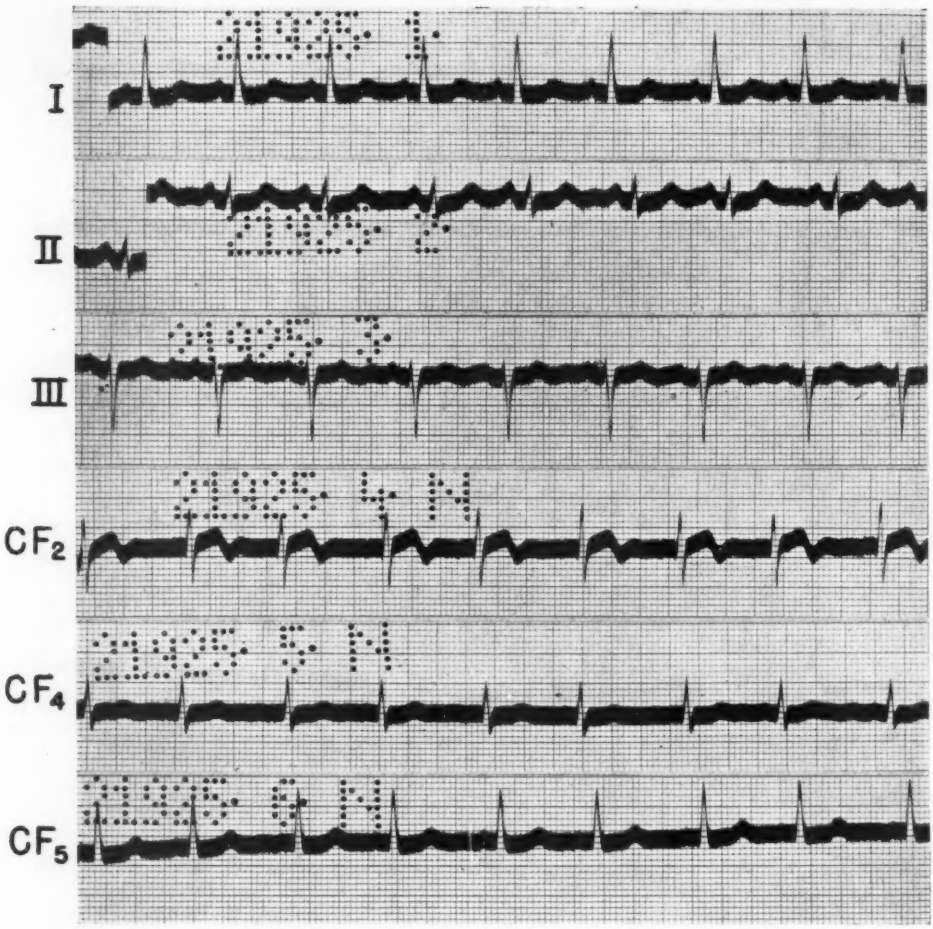


FIG. 2.—Premature systoles originating in the S-A node.

REFERENCES

Clerc, A., Levy, R., and Calo, A. (1938). *Arch. Mal. Coeur*, **31**, 1175.
Geiger, A. J., and Goerner, J. R. (1945). *Amer. Heart J.*, **30**, 284.
Lewis, T. (1925). "The Mechanism and Graphic Registration of the Heart Beat," 3rd ed., Shaw and Sons, London.
Scherf, D. (1945). *Amer. Heart J.*, **29**, 213.
Simon, A. J., and Langendorf, R. (1944). *Ibid.*, **27**, 345.
Wenckebach, K. F. (1908). *Arch. Anat. Phys.* (*Phys. Abt.*) **1**, 1907, and *III. Teil Suppl.*, 53, 1908.
—— and Winterberg, H. (1927). "Die unregelmässige Herztätigkeit." Wilhelm Engelmann, Leipzig.

DISSECTING ANEURYSM OF THE AORTA, WITH CARDIOGRAMS SUGGESTIVE OF CARDIAC INFARCTION

BY

GEOFFREY BOURNE AND P. J. W. MILLS

From St. Bartholomew's Hospital and the British Postgraduate Medical School

Received May 16, 1946

Two cases are here described in which the clinical picture was in some respects similar to that of myocardial infarction, and the electrocardiographic changes, although atypical, also suggested this diagnosis. Post-mortem examination showed the presence in each case of a dissecting aneurysm of the aorta, but revealed no evidence of any coronary infarction past or present.

The clinical pictures were suggestive of myocardial infarction in several respects. In each the pain was as severe as that seen in many cases of cardiac infarct, and its position although not typical was compatible with this diagnosis. In Case 1, the pain started in the front of the chest and was transmitted to the back; it also radiated to the upper abdomen. In Case 2, the pain was felt in the neck and through to the back under the scapulæ.

Shortness of breath was not a feature in either case, and was not complained of, apart from the distress naturally present before death. But shortness of breath is by no means constant in all cases of coronary infarction. Shock was a marked feature in both patients at some time; in Case 1 the systolic blood pressure fell to 80, and in Case 2 to 90 mm. In Case 2, pericardial friction developed and was sufficiently loud and characteristic to be demonstrated to students, although it was transient. In Case 1 the cardiogram was very suggestive of posterior infarct, and in Case 2 not only was well-marked S–T deviation present in leads II and IV, but the character of the tracing changed rapidly in two days, after which interval a bundle branch block had appeared.

It is interesting to speculate how these changes might have been produced; tabulation of various possibilities may be useful at this stage, to draw attention to these points in the details of the case reports.

1. Myocardial infarction might doubtless co-exist with a dissecting aneurysm.
2. The infiltration of the blood in the middle coat of the aorta might extend to a point where it influenced the coronary vessels passing through the aorta.
3. The infiltrating blood, in its way around the aortic curve, might stiffen the aorta, changing its normal tensions.
4. A similar process might twist it in the same way as the vessel is sometime twisted in pneumothorax, where cardiographic changes are also known to occur (Master, Dacks, Kalter, and Jaffe, 1937).
5. Finally, the ætiology of dissecting aneurysm is sometimes uncertain, but it seems that some degeneration of the substance of the aortic media may be a causative factor. It is theoretically possible that this degeneration may be acute, and may occur simultaneously in the substance of the heart muscle.

First Case Report

Case 1. A woman, aged 62, was admitted to hospital on 29/8/44 for diabetic symptoms with a six months' history of thirst, polyuria, loss of weight, undue fatigue, and paræsthesiæ in hands and feet. She also gave a history of an attack of very severe epigastric pain after a meal one week before admission; this pain radiated to the back, and was thought to be due to a flatulent dyspepsia although there was no such previous history.

Examination showed signs of recent loss of weight although the patient was still obese. Temperature, pulse, and respiration were normal. The retinal arteries were thickened, but the discs and retinæ were otherwise normal.

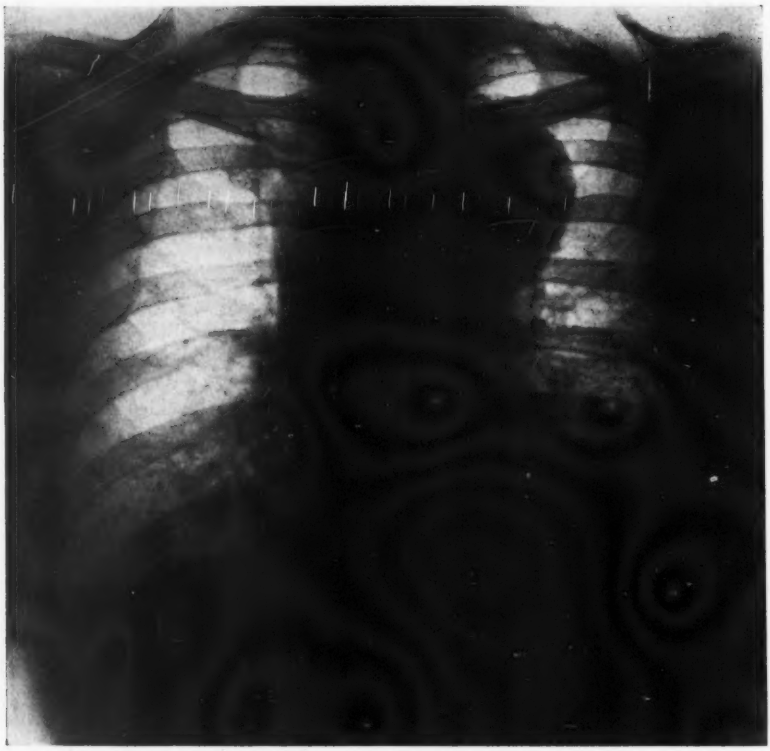


FIG. 1.—The left ventricle is enlarged. The aortic arch is enlarged and elongated, and projects unusually towards the left apical region. Pulsation was absent on screening.

There was a ringing aortic second sound. There was no clinical evidence of a mediastinal syndrome to suggest aneurysm. The B.P. was 220/120. The upper limbs were normal although the patient complained of tinglings in the fingers. The tendon reflexes were present and there was no sensory loss. The knee and ankle jerks were absent in both legs, and there was diminished sensation to pinprick, and impaired sensibility to light touch. Kinæsthetic sensation was present. The plantar reflexes were flexor.

In the urine (sp. gr. 1028), Benedict's solution was reduced to orange and Rothera's and Gerhardt's tests were positive. The fasting blood sugar was 278 mg. per 100 c.c. Sugar tolerance curve was typical of diabetes with blood sugar of 362 mg. per 100 c.c. two hours after 50 g. of glucose. Blood urea normal. Blood W.R. and Kahn negative. The cerebrospinal fluid was normal, and the W.R. negative.

X-ray of chest (Fig. 1) showed enlargement of left ventricle, aorta unfolded and elongated, with dilatation of the arch. No aortic pulsation. Lung fields clear. A diagnosis of diabetes mellitus with mild peripheral neuritis was made.

The diabetes was controlled by diet and 8 units of insulin b.i.d. and the fasting blood sugar was thus reduced to 120 mg. per 100 c.c.

On 2/9/44 (4 days after admission) the patient asked for a bed-pan and while on the pan complained of sudden intense tearing pain in the upper abdomen and middle of chest radiating to the back. She collapsed and became semi-comatose, with Cheyne-Stokes respiration. On examination she was very cold and the blood pressure fell to 80 systolic, the diastolic reading being unobtainable. Morphia was given with some relief of pain. After a few hours she made a gradual recovery, although the pain in the back persisted for the next two or three days, by which time she also had some difficulty in swallowing. A cardiogram taken 12 hours after the attack showed no changes suggestive of a coronary thrombosis (Fig. 2A).

On this history, especially the persistent pain in the back and the absence of the cardiographic changes of coronary thrombosis, a diagnosis of dissecting aneurysm of the aorta was made. During the next five days the patient's condition improved, the blood pressure gradually rose to 140/80, and the dysphagia disappeared. A further cardiogram (Fig. 2B) taken on 27/9/44 (five days after the attack) strongly suggested a posterior myocardial infarction as shown by a deep Q III and elevated S–T and inverted T, both in lead III. It was then thought that the original diagnosis of dissecting aneurysm was wrong and that a coronary thrombosis must be the cause of the pain and collapse. A further cardiogram (Fig. 2C) still suggested myocardial infarction.

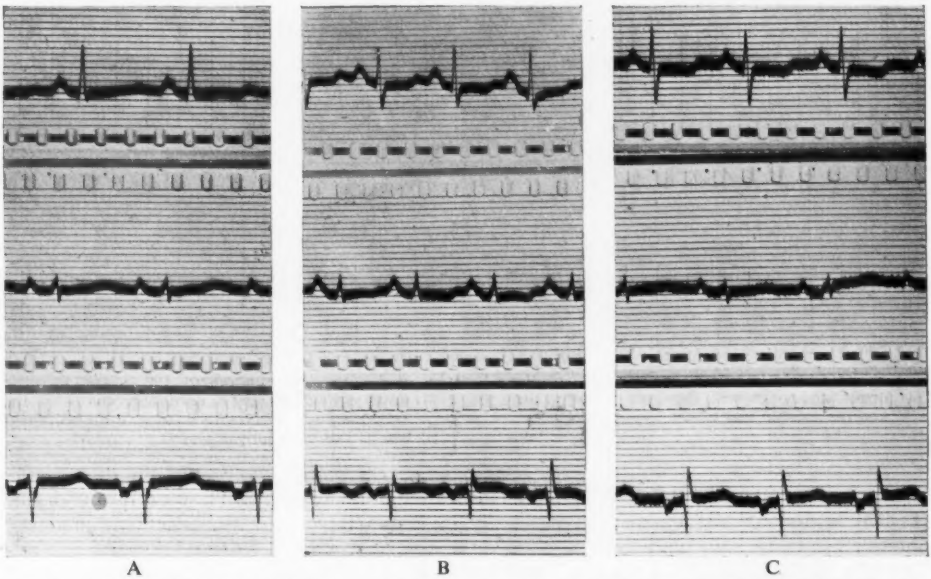


FIG. 2.—Left axis deviation. (A) 2/9/44. P wave sharply peaked in leads I and II and inverted in III. No evidence of myocardial infarct.
(B) 27/9/44. S–T deviation downwards in leads I and II and upwards in III. Deep Q in lead III. T now inverted in lead III.
(C) 11/10/44. Similar to Fig. 2 (B). T wave further inverted in lead I.

On 14/10/44 the patient suddenly had a severe hæmoptysis; she collapsed and rapidly became comatose, the blood pressure falling to 60 systolic. She remained comatose for about three hours and during this time passed a tarry stool and then died. In view of the terminal hæmoptysis and melæna, the original diagnosis of dissecting aneurysm of the aorta was thought to be established, although this made the cardiographic changes difficult to explain.

Post-mortem Examination

Pleural spaces dry, lungs expanded, two large thoracic dissecting aneurysms, loaded abdominal fat depots, small intestine distended with blood.

Heart and Aorta. Heart: 280 g., valves and chambers normal, coronary arteries atheromatous but nowhere occluded or grossly narrowed. Aorta: ectasia and fatty atheroma of ascending portion, severe calcified atheroma of arch and descending thoracic and abdominal portions. Junction of transverse and descending aorta marked by beginning of a dissecting aneurysm localized to medial wall and bulging into subepicardial portion of left lung (8 cm. long, 4 cm. wide, 3 cm. outward bulge into lung) (Fig. 3). No obvious primary intimal tear; instead there was a large patch proximally of ulcerated intima covered by laminated clot. Externally, this part of aneurysm was firmly attached to the lung by fibrous adhesions and organized thrombus. It contained a small linear longitudinal external tear through which a probe passed from the aortic lumen direct into upper lobe of left lung, which was torn up by

FIG. 3.—Posterior aspect of thoracic aorta showing the upper aneurysm with laminated clot at its apex, and the intimal tear of the lower aneurysm.

FIG. 4.—Transverse section of the lower thoracic aneurysm showing compression of the oesophagus. Blood can be seen in the sectioned bronchi of both lungs.

hæmorrhage. 10 cm. distal to the isthmus the intima presented a transverse rupture of 1 cm. (Fig. 3) leading into the proximal portion of a second large dissecting aneurysm, bulging anteriorly and laterally into lower lobe of right lung (8 cm. long, 4 cm. wide, 5 cm. bulge). This was firmly adherent to the lung, and there was no external tear. The left auricle was pushed forwards and the œsophagus compressed anteriorly and to the right (Fig. 4). Blood clot in both aneurysms was lying inside the split medial coat of the aorta. Transverse sections of the abdominal aorta showed two separate incipient aneurysms in the form of hæmatomas of the media (each about 1 cm. in diameter) lying 5 and 9 cm. distal to the coeliac axis (Fig. 5). The overlying intima was intact.

The trachea and bronchi and their branches in both lungs were filled with blood. Lung parenchyma: mixed hæmorrhage and emphysema. Pool of blood in nasopharynx. Stomach: distended with 500 c.c. mixed blood and gastric juice; no peptic ulceration. Intestines: contained blood and tarry material down to rectum. Viscera pale; arteries atherosclerotic.

Death was due to hæmorrhage from rupture of an aortic dissecting aneurysm into the left lung. Blood had entered the air-passages (precipitating an acute emphysema) welled up into nasopharynx and passed down into stomach during patient's terminal coma.

Microscopic Findings. Aorta shows atrophy and diminution in number of medial elastic fibres, fatty degeneration of internal elastic lamina, localized complete medial atrophy underlying some intimal atheromatous plaques, no mucoid or cystic degeneration but marked increase in hyaline and collagen at junction of outer and middle thirds of media (surrounding most internal branches of vasa vasorum) (Fig. 6). It is this part of the middle coat that is split to enclose the hæmatomas and aneurysms. The plane of cleavage in the upper aneurysm is filled with young granulation tissue rich in phagocytes loaded with blood pigment (Fig. 7). The local adventitia shows vasa vasorum cuffed with lymphocytes, histiocytes, and pigment phagocytes. No syphilitic mesaortitis. The upper aneurysm appeared to be the oldest lesion; the outer wall consisted of fibrosing granulation tissue rich in hæmosiderin.

FIG. 5.—Transverse sections of the medial hæmatomas (incipient aneurysms) in the abdominal aorta.

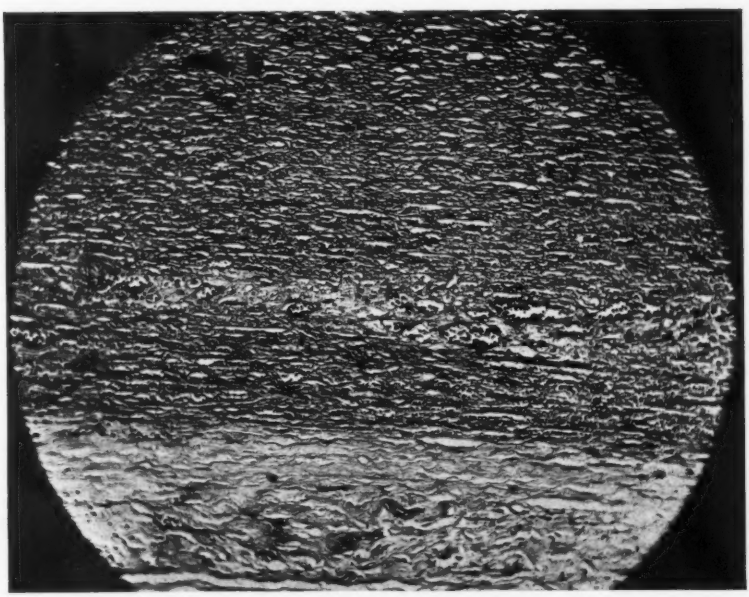


FIG. 6.—Section of thoracic aorta media (iron hæmatoxylin, van Gieson and Verhoef): atrophy of elastic fibres with hyalinization and increased vascularity at the junction of the outer with middle third of the media. Magnification: × 97.

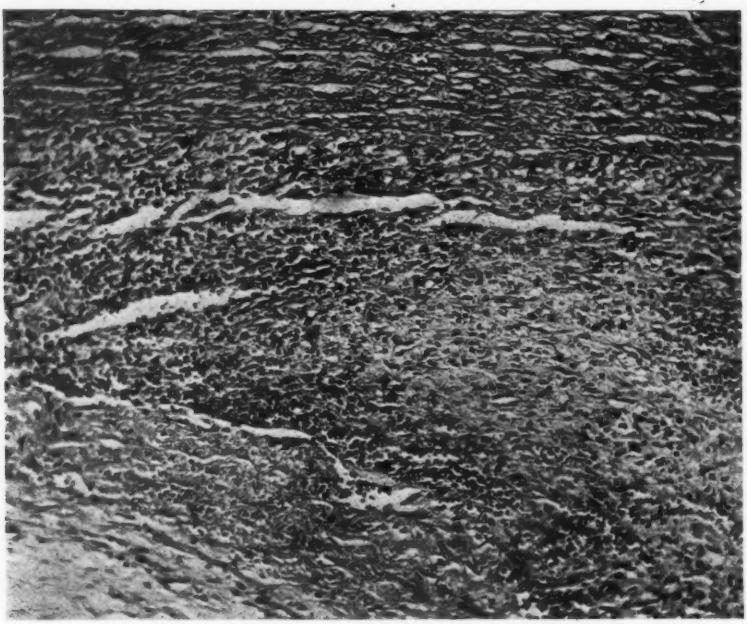


FIG. 7.—Section of edge of upper aneurysm (hæmatoxylin and eosin): granulation tissue separating the outer third of the elastic media from the inner two-thirds. Magnification : × 130.

The *heart* shows very little increase in interstitial fibrous tissue except for subepicardial pale areas in the left ventricle which consist of ancient acellular collagen.

The sequence of events appears to have been first a chronic degenerative hyalinization of the middle coat of the aorta, secondly acute hæmatoma formation in the media at the junction of middle and outer thirds, thirdly rupture of the underlying intima, and fourthly dissection of the vulnerable media by the inrushing aortic blood with the formation of dissecting aneurysms. In this case there is no doubt that atheroma has played an important role in the localization of the site of primary intimal rupture.

SECOND CASE REPORT

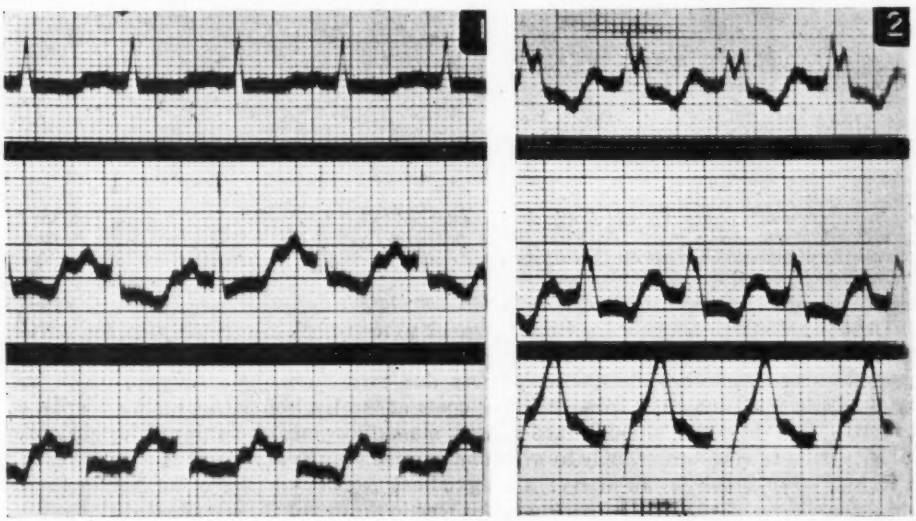
A housewife, aged 36, had for a period of ten weeks before admission been complaining of pain below both shoulder blades and upon each side of the neck. She suffered from this pain in attacks which would pass off, and they might be associated both with nausea and with giddiness. She was not short of breath in the intervals between the attacks, but noticed some dyspnœa at the time. The original attack occurred while she was ironing, and the pain and discomfort were severe enough to need the attention of a doctor. This attack lasted throughout the day and increased in severity until she vomited in the evening. Since the first attack she had felt what she described as a lump in her throat, which was present all the time and especially noticeable on swallowing.

Five weeks before admission, on getting up in the morning she had a particularly severe attack associated with intense nausea. She recovered, however, and continued up and about, feeling fairly well except for slight recurrent attacks of the nature described above. She had no cough, no pain in the front of the chest, no hæmoptysis nor interference with breathing. Her appetite had been good and there were no other complaints. On the morning of admission she was seized with a more severe attack than she had previously suffered, and was admitted lying flat on a stretcher, intensely cyanosed, with a raised respiration rate and grunting expiration. There was no stridor. She also complained of numbness in the left arm, and kept her eyes closed because of intense vertigo.

On examination, which was limited in thoroughness by the patient's very distressed condition, the following outstanding points were noticed at this time. There was no orthopnœa. Cyanosis was intense; arterial and venous pulsation in the neck vessels was more than normal. The apex beat was outside the mid-clavicular line and was firm and normal in character. There were no murmurs. There appeared to be some slight diminution in the volume of the respiratory murmur at the right apex, and râles were present over the right chest. In other respects the lungs and pleuræ seemed to be normal. Later in the day the patient became extremely short of breath with increased cyanosis and with stridor. She was very restless. She then spat up a quantity of thick, frothy, unstained sputum, and was given morphia 1/6th, and atropine 1/100th of a grain, and oxygen by B.L.B. mask. She improved during the next 48 hours. Two days after admission the increased pulsation was still visible in the neck, the heart sounds were still normal, but in addition a triple friction sound was audible all over the præcordium, and especially in the area between the apex beat and the left sternal border. The blood pressure was 94/40, the heart rate 112, and the respiration rate 40.

During the day the patient became comatose and died.

The electrocardiogram taken on the day of admission (Fig. 8A) showed flattening of T I and well-marked S–T deviation downwards in leads I, II, III, and IV. The tracing taken two days later (Fig. 8B) showed the development of a left bundle branch lesion. The X-ray



A B

FIG. 8.—(A) Taken on day of admission, showing S–T deviation downwards in leads I, II, and III. T flat in lead I. (B) Two days later, showing left bundle branch defect.

film (Fig. 9) showed deviation of the trachea to the right at the level of the sterno-clavicular joints. Behind the left joint was a shadow corresponding to the post-mortem demonstration of an extension of the dissecting aneurysm along the origin of the left subclavian artery. The right lung was less translucent than the left, and the radiologist suggested the possible presence of some scattered consolidation, possibly in relation to an infarct. The right pulmonary artery was very prominent. These radiological differences between the two sides of the chest corresponded to the difference in physical signs. Such change could easily be produced by central mediastinal changes causing interference with the pulmonary arterial or venous systems. The heart showed no radiological evidence of increase in size.

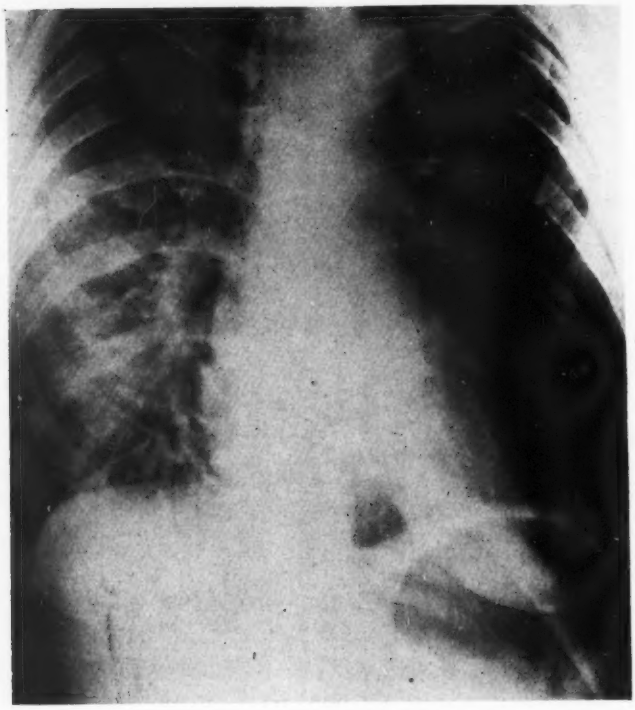


FIG. 9.—Heart within normal limits. Aortic shadow extended to left subclavicular region, corresponding to site of subclavine extension of aneurysm. Right pulmonary artery abnormally prominent. Increased vascularity of middle and lower zones of right lung.

Post-mortem Examination

The body was well nourished. Head and neck were normal.

Thorax. On drawing forward the mediastinal contents the aorta was seen as a thickened, shaggy-looking structure. The thickening was due to a dissecting aneurysm, which had completely stripped the outer from the inner coats at the level of the ascending portion of the aorta. The dissecting blood formed a larger mass of organizing clot, probably six weeks old; this mass extended proximally to within half an inch of the aortic valves and also along an inch of the left subclavian artery; a longitudinal slit, one-quarter of an inch in length with sharp edges as though cut with a knife, was seen in the intima; it was situated at the distal end of the solid organizing clot, i.e. at the junction of the ascending and transverse arches of the aorta. The dissecting mass of fluid blood extended to the aortic bifurcation. Distal to the slit in the intima wall the dissecting blood was dark and fluid; it could be pressed back through the slit. The aortic lumen was reduced to the size of a crow quill.

Heart. The heart was not enlarged. The coronary arteries were not involved in the aneurysm. Dr. Discombe stated that atheromatous plaques did not involve the mouths of the coronaries but were present along their lengths. There was no evidence of any coronary infarction. Terminal focal necrosis of a marked degree was present. It is possible that in the intact organ the tumour formed by the extravasated blood could have pressed on the coronary arteries although it did not directly involve them. This pressure would have interfered more with the left coronary and the anterior descending branch. Myocardium, normal macroscopically. A minimal quantity of clear pericardial fluid present. Surfaces normal. Valves normal. Aorta, no atheroma, intima healthy.

Lungs both oedematous. Liver congested; stomach congested slightly; otherwise no abnormality of the gut.

Microscopically the heart muscle showed focal necrosis. The affected fibres were swollen and had lost their transverse striation and were more deeply eosinophilic than normal. Nuclei had disappeared and there were small numbers of polymorphs infiltrating the necrotic areas and an increase in epimysium cells. Dr. Discombe stated that the focal necrosis in the heart was most remarkable.

Discussion

There would thus appear to be no clear correlation between the post-mortem findings and the electrocardiographic changes. In neither case was there any evidence of occlusion or gross narrowing or infarction of the coronary circulation. In Case 1, both aneurysms were situated in the descending aorta. It was impossible to determine from the post-mortem reports of either case whether the presence of the dissecting aneurysms could have displaced or twisted the heart in such a way as to interfere with the coronary flow, but the radiological evidence in Case 2 did not support this hypothesis. In each case the cardiogram was very suggestive of posterior coronary infarction: in Case 1 there were changes in the ten days interval between the first and second tracings that strongly favoured a coronary occlusion; in Case 2 the bundle branch tracing that developed may have been a manifestation of the dying heart, since it was present on the day the patient died, but the previous curve showing S–T depression was taken two days before the patient died.

Unfortunately few electrocardiograms have been studied in dissecting aneurysm of the aorta because the patient so frequently dies before one can be taken. In 19 cases studied by Glendy, Castleman, and White (1937), only 2 had cardiograms taken; one of these was normal and the other showed changes suggestive of posterior coronary infarction, but in this instance it was found post-mortem that the aneurysm involved the mouths of the coronary arteries. This was not so in our two cases.

If the changes were due to the great lowering of blood pressure only (Katz, 1941), they should have been present in the cardiogram taken twelve hours after the second severe attack, when the blood pressure was at its lowest: but they were not present. The fibrous patches in the left ventricle in Case 1 were seen on section to be acellular and, therefore, of considerable standing; but in Case 2 the myocardial necrosis may have been significant. If these three possible causes are ruled out, it is difficult to explain the cardiographic changes.

In Case 1, the first attack of pain radiating to the back probably occurred at the time of formation of the upper aneurysm; the second, with characteristic tearing pain, persistent pain in the back, and subsequent dysphagia can be well correlated with the anatomical findings of the lower aneurysm of the thoracic aorta. It was this clinical picture that supported the correct diagnosis after the second attack, especially in view of the persistent pain in the back, an unusual symptom in coronary thrombosis. This case is also atypical in the site and multiplicity of its aneurysms.

The pathological findings in Case 1 strongly suggest that the aneurysms were preceded by hæmatomas produced by rupture of vasa vasorum into a media deficient in elastic fibres. A greatly increased vascularity of the wall of atheromatous vessels with a tendency to intramural hæmorrhage has been well described by Winternitz, Thomas, and LeCompte (1938). The majority of dissecting aneurysms arise in the ascending aorta and, in the absence of local atheroma, upon a basis of cystic medial necrosis coupled with hypertension; it does thus appear that in the descending aorta atheroma, by its associated increased mural vascularity, may play an important if indirect part in the pathogenesis of medial hæmatomas. In addition, advanced atheroma leads to pressure atrophy of the media, and finally may also determine the site of primary intimal rupture.

Summary and Conclusions

The most striking feature, and one not previously described, is the misleading influence of the electrocardiogram in the diagnosis of these two cases of dissecting aneurysm. The changes in it, when associated with the rest of the clinical picture, were sufficiently typical of myocardial infarction to mislead attention from the correct diagnosis. Thus the assumption that such changes are absent in dissecting aneurysm may be false.

The most important points in the differential diagnosis would seem to be as follows. The pain in both cases was referred to the back to a considerable extent, this pain was periodic and extended over a period of as long as 15 weeks in Case 2, with periods of remission. Absence of shortness of breath in an ambulatory patient would also be an important point. Evidence of mediastinal mechanical change should also arouse suspicion of a dissecting aneurysm. The numbness in the left arm and the lump in the throat in Case 2, and the dysphagia in Case 1, all bring out this point. The severity of the pain is such that few conditions other than coronary infarction are likely to be suggested. A pain of similar severity may occur in acute pancreatitis, but here it is the lower part of the back rather than the interscapular area that is the site of the pain.

The post-mortem investigations and notes in Case 1 were the work of Dr. I. Doniach of the British Postgraduate Medical School, Hammersmith; those of Case 2 were the work of Dr. George Discombe of St. Bartholomew's Hospital.

We are grateful to them for permission to publish these; and express our thanks to them for their help, and also to Dr. McMichael of the British Postgraduate Medical School for his kind co-operation in Case 1.

References

Glendy, R. E., Castleman, B., and White, P. D. (1937). *Amer. Heart J.*, **13,** 129.
Katz, L. N. (1941). "Electrocardiography," Philadelphia, pp. 162, 251.
Master, A. M. Dacks, Kalter, H. H., and Jaffe, H. L. *Amer. Heart J.* (1937), **14,** 297.
Winternitz, M. C., Thomas, R. M., and LeCompte, P. M. (1938). "The Biology of Arteriosclerosis," C. Thomas, Springfield, Ill., U.S.A., p. 87.

CHRONIC DISSECTING ANEURYSMS

BY

A. MORGAN JONES AND F. A. LANGLEY

From the Cardiographic Department, Manchester Royal Infirmary, and the Department of Pathology, University of Manchester

Received May 20, 1946

In 1939 East was able to find records of only 27 instances in which the diagnosis of dissecting aneurysm had been made during life. Usually death from complete rupture of the aorta quickly follows the initial dissection; of the 300 cases collected by Shennan (1934) 210 died within a week and survival for more than a year was recorded in only 16 cases. We have, therefore, thought it desirable to record two cases in which the diagnosis was made during life; one patient survived for three years and the other is alive and comparatively well eight years after radiographic recognition of the aortic lesion.

Case Records

Case 1. In December 1943, a married woman, aged 55, was admitted to the Manchester Royal Infirmary under the care of Dr. Crighton Bramwell, on account of severe pain in the back of the chest and in the lumbar area. Apart from an attack of smallpox when 4 and an operation for uterine prolapse when 41, she enjoyed good health until the age of 52 years. She was then suddenly seized by severe stabbing pain in the left anterior chest which radiated to the left scapular region and the dorso-lumbar area. She fell to the ground but did not lose consciousness. She was confined to bed for two weeks and suffered from some aching pain in the left chest for four or five weeks. Subsequently she remained well for nearly a year, when a similar attack took place; her blood pressure was then found to be 230/130. Following this attack her pain again gradually disappeared and she remained in comparatively good health for a year, when her third attack of severe pain occurred. She was admitted to hospital and the heart and aorta were found to be enlarged radiographically. On this occasion, however, her pain did not subsequently disappear, but extended to the lumbar area. After several months of persistent pain in the chest and back she was admitted to the Manchester Royal Infirmary in April 1943, under the care of Dr. Oliver. Her blood pressure was then 210/135 in the right arm and 195/130 in the left. Cardioscopy showed gross dilatation of the aorta. The Wassermann reaction was negative and a diagnosis of chronic dissecting aneurysm was made. She remained in bed for four months, but was never free from pain. When she got up, walking 20 yards brought on severe anginal pain; this gradually improved, but in November 1943 she began to suffer from attacks of palpitation of sudden onset, lasting several hours. She was readmitted under the care of Dr. Bramwell. Her blood pressure was then 225/140 in the left arm and 210/140 in the right. On cardioscopy the aorta was grossly dilated and the left ventricle enlarged (Fig. 1 and 2). The blood Wassermann and Kahn reactions were negative. A cardiogram showed left axis deviation and biphasic T waves (− +) in leads I and II, with depression of the corresponding RS–T segments; these appearances were attributed to left ventricular strain associated with her hypertension. While in hospital she had several attacks of palpitation, during one of which a cardiogram showed paroxysmal auricular tachycardia at a rate of about 160 a minute. These attacks could be stopped at once by carotid sinus pressure, but the ectopic rhythm was re-established a few minutes after pressure was released. While in bed her pain diminished but did not altogether disappear. Some eight weeks after admission, and shortly before she was to go home, she collapsed in the lavatory and fell to the ground semi-conscious, with an imperceptible pulse. She improved slowly but complained of severe pain in the left chest; four days later she suddenly collapsed and died within a few minutes.

A *necropsy* was performed nine hours after death. Apart from slight passive congestion of the abdominal viscera, the abnormalities were confined to the thorax. The left pleural cavity was filled with straw-coloured fluid and blood clot; this had led to collapse of the left lung.

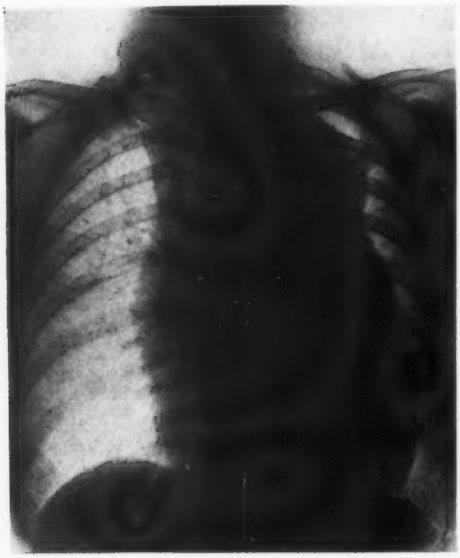


FIG. 1.—*Case* 1. Postero-anterior teleradiogram. The heart outline can be seen in front of the shadow of the greatly enlarged aorta.

FIG. 2.—*Case* 1. Left anterior (II) oblique radiogram, showing the great size of the descending thoracic aorta. The aortic window is still visible for the ascending aorta is not greatly enlarged.

The heart weighed 390 g., the left ventricle was hypertrophied but not dilated, and there were many pearly-grey areas of myocardial fibrosis. The right ventricle and all the valves were normal. The circumference of the aortic orifice was not increased (6 cm.). Numerous atheromatous plaques were present in both coronary arteries but the lumen was not severely narrowed at any point.

Externally the aorta was greatly dilated from just below the origin of the left subclavian artery to the level of the diaphragm (Fig. 3). At about the middle of the lateral surface of the dilated portion there was a transverse tear, 2 cm. long, where the aneurysm had ruptured into the left pleura. On opening the aneurysm (Fig. 4) two channels were seen; on the inner border a narrow, flattened channel (A) led from the normal arch of the aorta to a dilated blind sac, in which the orifices of the last two pairs of aortic intercostals could be seen; this narrow channel was, therefore, the compressed descending thoracic aorta. The second channel (B) formed the greater part of the specimen and was continuous with the arch of the aorta above and with the abdominal aorta below. It was partially lined by laminated thrombus (C) and the external tear communicated with it. Where the two channels arose from the arch of the aorta the free edge of the septum separating them was rounded and smooth and continuous with a ridge around the mouth of the larger channel (Fig. 4, inset); the free margin had clearly been separated from this ridge when the dissection started and, since both ridge and free edge were smooth and healed, this separation was not recent.

Sections from the arch of the aorta, the walls of the aneurysm, and the abdominal aorta were stained with hæmalum and eosin, van Gieson, mucicarmine, Sudan IV, and Weigert's elastic stain. Sections from the aneurysm showed that at a and a′ (Fig. 5, see p. 195) the media had split, two-thirds of its tissue passing inwards to cover the septum between the two channels and one-third lining the outer wall of the larger channel; the aneurysm had thus arisen by a separation of the outer third from the inner two-thirds of the media. Within the intima of the original aorta (A), at its acute angles (near a and a′), there were polyhedral cells loaded with fat and between them large blood capillaries, while superficially a zone of young connective tissue and organizing blood clot invaded the original lumen of the aorta; in time this process could have obliterated the lumen of the compressed aorta. The lateral wall of the aneurysm was lined by dense fibrous tissue covered in some places by endothelium, in others by organizing thrombus.

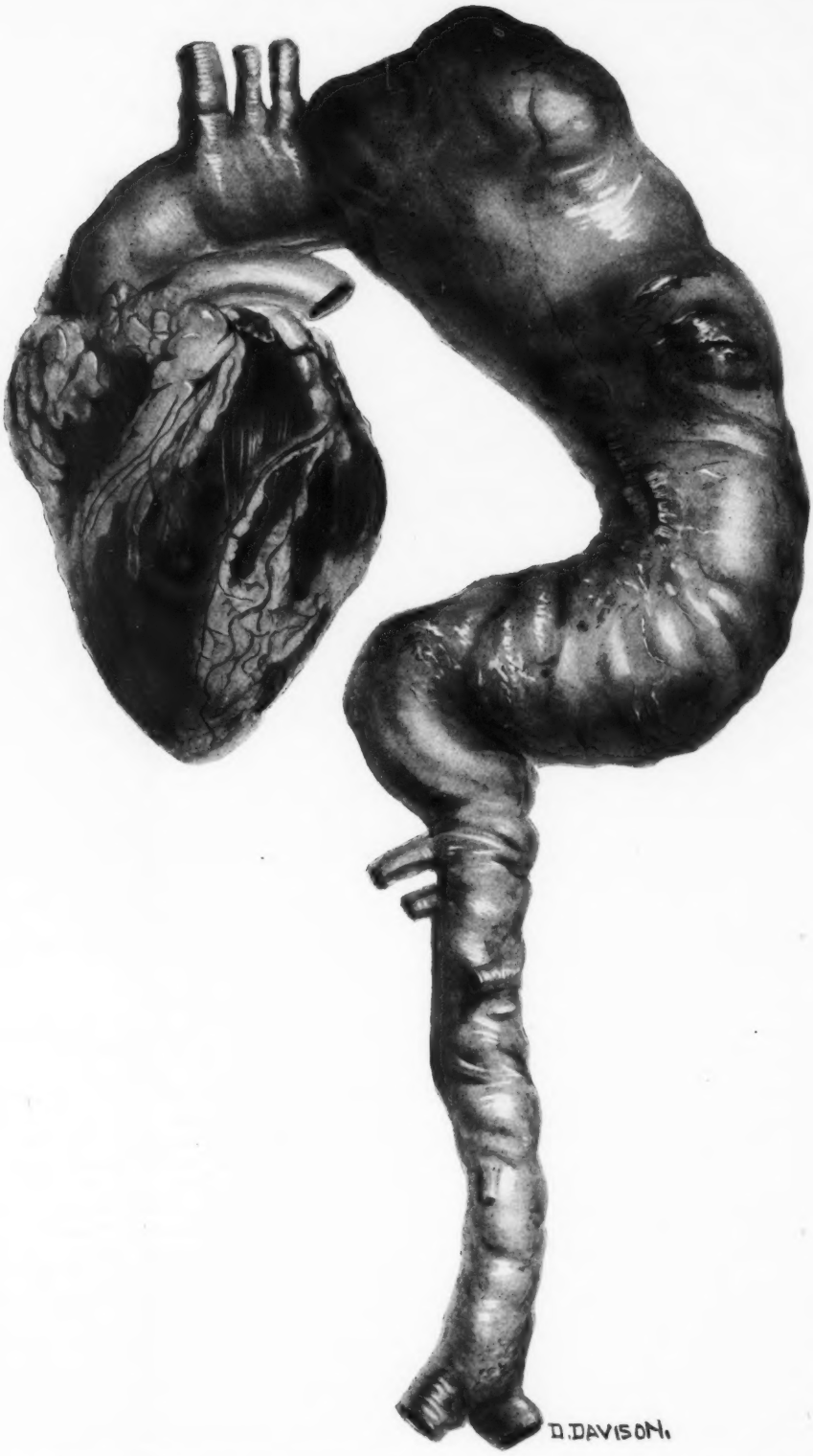


FIG. 3.—*Case* 1. The aneurysm from a water-colour painting. The transverse tear is visible on the left border of the aneurysm. The aneurysm does not extend below the diaphragm. The ascending aorta and the abdominal aorta are not enlarged.

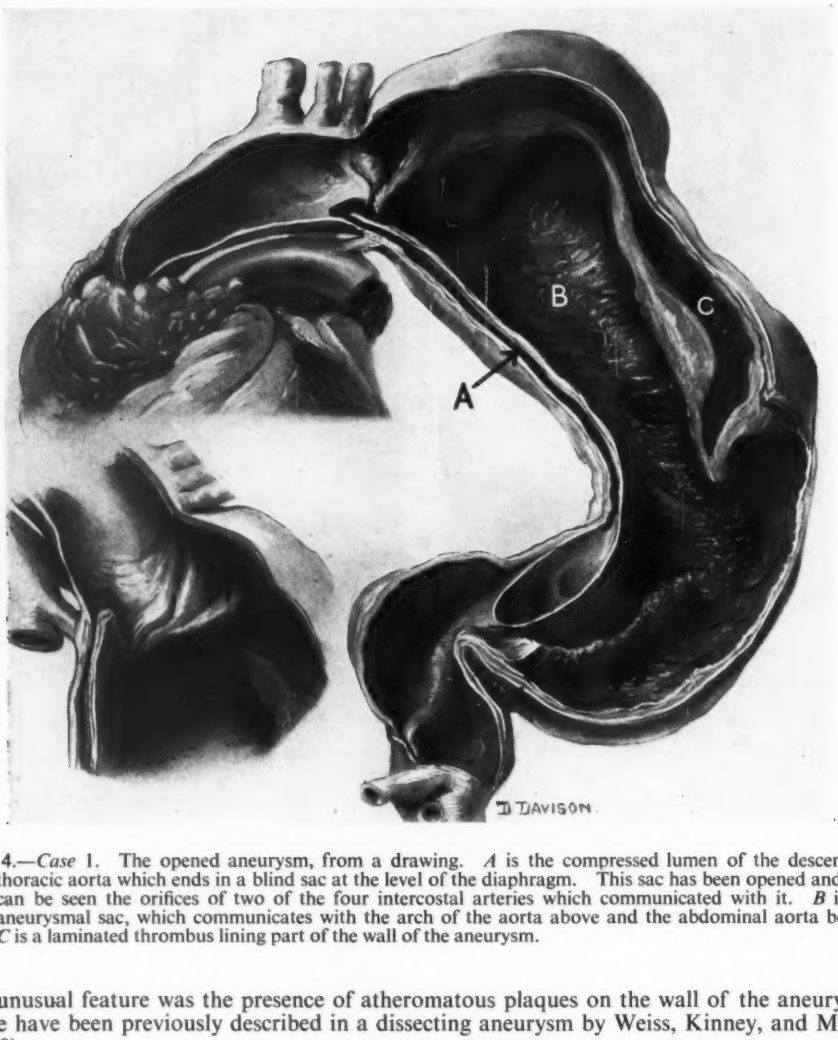


FIG. 4.—*Case* 1. The opened aneurysm, from a drawing. *A* is the compressed lumen of the descending thoracic aorta which ends in a blind sac at the level of the diaphragm. This sac has been opened and in it can be seen the orifices of two of the four intercostal arteries which communicated with it. *B* is the aneurysmal sac, which communicates with the arch of the aorta above and the abdominal aorta below. *C* is a laminated thrombus lining part of the wall of the aneurysm.

An unusual feature was the presence of atheromatous plaques on the wall of the aneurysm; these have been previously described in a dissecting aneurysm by Weiss, Kinney, and Maher (1940).

In the arch of the aorta there were striking medial degenerative changes for many small cyst-like spaces were seen and an excess of mucin was present, particularly around the cysts. Similar, though less striking, changes were also found in the media of the wall of the aneurysm, but they were not present in the abdominal aorta. The medial degeneration was thus most striking proximally, unlike the atheromatous changes which were very slight in the arch but of moderate severity in the abdominal aorta. There was no evidence of destruction of elastic tissue apart from a few complete breaks at the angles of the compressed aorta. The adventitia and vasa vasorum were normal and there was no suggestion of syphilis of the aorta.

The aneurysm appeared to have developed from the entry of blood into the media just beyond the origin of the left subclavian artery; the media was split at the junction of its outer third and inner two-thirds and the dissection extended until the diaphragm obstructed its advance. The pressure in the aneurysmal sac led to compression of the original aortic lumen and to the re-entry of the aneurysm into the aorta below the compressed portion. Thus the blood supply to the legs and abdomen, transiently interrupted by occlusion of the aorta, was restored by re-entry of the aneurysm and the aneurysmal sac took up the original function of the aorta. Healing of the aneurysm followed; first by organization of the clotted blood lining

its walls, then by the development of a lining of endothelium. Finally, reparative processes began in the angles of the compressed aorta; this process, if continued, would have led to obliteration of the functionless lumen.

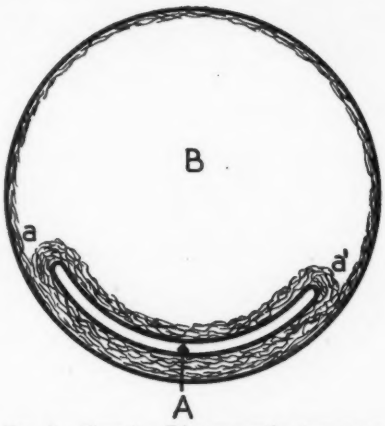


FIG. 5.—*Case* 1. Diagrammatic transverse section of the aneurysm. At a and a′ the media has split, one-third of its tissue lining the outer wall of the aneurysmal sac (B), and two-thirds covering the septum between the aneurysmal sac and the compressed lumen of the aorta (A). The aneurysm was, therefore, formed by splitting the media.

Case 2. In 1939, a single woman, aged 36 years, was admitted to the Manchester Royal Infirmary under the care of Dr. Crighton Bramwell, complaining of palpitation and breathlessness. Apart from measles as a child, she had been well until 1933, when she was a probationer nurse, aged 30 years. She then began to suffer from severe pain in the back which was treated by radiant heat and massage and disappeared after several months. In January 1938 she developed acute bronchitis; during this illness she awoke one night with a severe attack of palpitation and subsequently suffered from dyspnœa, palpitation, and lassitude. Some weeks later chest radiograms were taken and an enlarged substernal thyroid was diagnosed; she was treated with Lugol's iodine without benefit. In April 1938, while on sick leave in Ireland, she was admitted to hospital on account of her dyspnœa and palpitation. Her blood pressure was found to be 200/140 and radiograms of the chest showed moderate cardiac enlargement and considerable dilatation of the aorta. She was treated by X-ray therapy to the sternal area, but this was followed by intense pain in the back. While in hospital she had a uterine hæmorrhage and underwent a myomectomy for uterine fibroids; after operation she had periods of mental confusion and amnesia, and severe paroxysms of palpitation. In September, while trying to make her bed, she suddenly collapsed, and in December 1938, developed sudden severe dyspnœa with evidence of circulatory collapse. In March 1939 she returned to Manchester and remained in bed until she was admitted to hospital in May owing to a severe attack of constricting chest pain which was followed by vomiting. Subsequently she had three similar attacks and in September developed acute bronchitis with hæmoptysis. A fifth attack of constricting chest pain followed this illness and in October 1939 she was admitted to the Manchester Royal Infirmary.

On examination the pulse rate was 120 and her brachial blood pressure 200/140; the same reading was obtained in the legs and there was good pulsation of the dorsalis pedis arteries. The heart sounds were very loud but no murmurs were present. Cardioscopy showed some enlargement of the left ventricle and considerable diffuse enlargement of the thoracic aorta (Fig. 6) with calcified plaques in its wall (Fig. 7). Kymography revealed quite good pulsation of the descending aorta. A cardiogram showed striking left axis deviation associated with biphasic T waves (− +) in leads I and II and depression of the corresponding RS–T segments; these appearances were attributed to left ventricular strain associated with her hypertension. The blood Wassermann and Kahn reactions were negative and the urea clearance 75 per cent of the average normal. The condition was thought to be a chronic dissecting aneurysm.

While in hospital she suffered from severe aching pain in the left scapular region, headache, and palpitation. On several occasions she became confused and disorientated. Her condition had slightly improved when she left hospital in January 1940, but she continued to suffer from pain in the back and headaches until she was readmitted in December 1940, owing to the increasing severity of her pain and the onset of aching pain in the left leg. After her return home she remained in fair health, apart from the pain, until April 1941 when her right arm and leg became paralysed while she was out walking. The paralysis rapidly improved but some weakness of the right arm remained. In May she had a severe attack of sharp pain in the back of the chest, just to the left of the spine and in the left infra-scapular area. Subsequently she suffered from constant aching pain in this area with recurrent severe attacks of sharp pain, often precipitated by exertion. In August, severe aching pain developed in the epigastrium, and in November 1941 she was readmitted to hospital owing to the severity of her epigastric and chest pain. Her brachial blood pressure was then 165/110 and the dorsalis pedis pressure 205/110. While in hospital she had two sudden severe attacks of

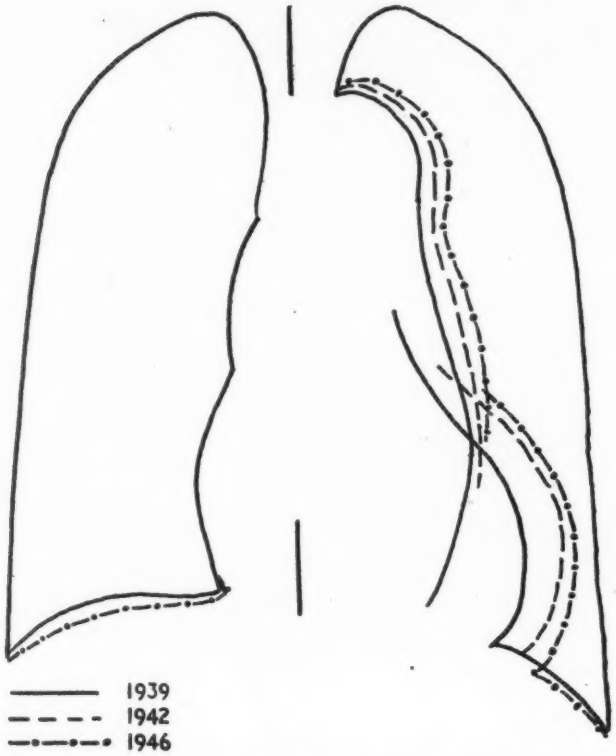


FIG. 6.—*Case* 2. Outline drawings from postero-anterior teleradiograms, 1939, 1942, and 1946. The chest outlines were almost identical in all three radiograms and there has been little change in the contour on the right side of the cardiac silhouette. The gradual enlargement of the left ventricle and of the aorta is shown.

" grinding " pain in the left chest, relieved only by morphia, and followed by nausea and vomiting.

After her return home the pain continued to increase in severity and by March 1942 had become intolerable. When she was readmitted to hospital she complained of constant aching pain in the lower costal and lumbar region with several severe exacerbations each day, relieved only by morphia, and sudden attacks of constricting sternal pain, terrifying in character, but unrelated to exertion. The severity of these pains and the gradual deterioration in her condition demanded radical measures and paravertebral proctocaine injection of the upper 8 dorsal interspaces on both sides was attempted after a successful novocaine test. Unfortunately, the proctocaine injection failed to give much relief, and in August, Mr. Geoffrey Jefferson undertook paravertebral alcohol injection. After operation her pain remained severe for some weeks and she developed hyperæsthesia of the lower left thoracic root areas, fæcal and urinary incontinence, and pyramidal signs. Nevertheless, her pain gradually diminished and a year later, in August 1943, she was almost entirely free from pain and her neurological signs and symptoms had completely disappeared.

In 1944 she suffered from depression and was reluctant to get out of bed, but during the whole of 1945 she was active, undertook a great deal of housework and cooking and went to dances; she was entirely free from pain, gained weight, and lost all interest in her illness. In December 1945, however, cardioscopy showed some increase in the size of the heart and aorta and in the calcification of the aortic wall (Fig. 6, 7, and 8). Lateral and oblique radiograms of the thoracic spine did not show any erosion, the blood Wassermann reaction was negative, and the urea clearance 74 per cent of the average normal. It was noted that the right radial pulse was poorer than the left; the blood pressure in the right brachial artery was 200/125, in the left 215/125, and in the left dorsalis pedis artery 240/125.

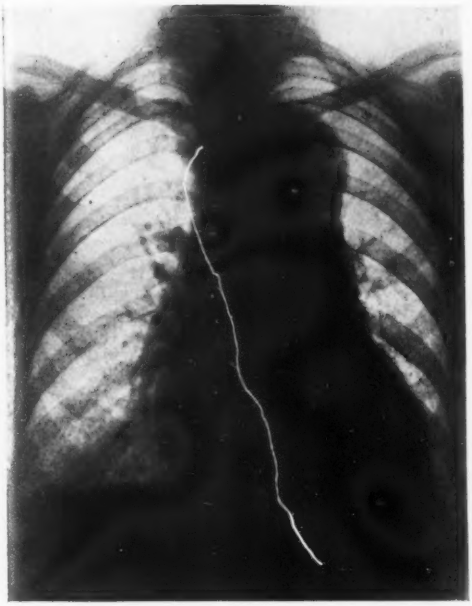


FIG. 7.—*Case* 2. Postero-anterior teleradiogram, December 1945. Calcification can be seen in the enlarged aortic knuckle.

FIG. 8.—*Case* 2. Right anterior (I) oblique radiogram showing calcification of the enlarged aorta.

In January 1946 she was readmitted with acute bronchitis, from which she made an uneventful recovery.

DISCUSSION

Virchow and other pathologists of his time thought that dissecting aneurysms arose from the dissection of blood through the floor or edge of an atheromatous ulcer. Subsequently this opinion was criticized by many pathologists and Shennan (1934) has reviewed the subject fully. In his own series Shennan found that only 6 of 218 recent dissecting aneurysms appeared to have started from an atheromatous ulcer: he emphasized the importance of medial degenerative changes, particularly those involving the elastic laminæ, and believed that the media nearly always gave way before the intima. Rottino (1939) studied the distribution of medial degenerative changes in 12 dissecting aneurysms and found that these changes were confined to the ascending and transverse aorta in 11 cases; this is probably significant for it is in these sites that the majority of dissecting aneurysms begin. Sailer (1942) studied and classified the types of medial degeneration that occur in the aorta. The histological appearances in our Case 1 are in general agreement with the views expressed by Shennan (1934) and Rottino (1939), and the degeneration is similar to that in certain of the types described by Sailer (1942).

Since survival for more than a few days is the exception when the aortic media is shattered by a sudden hæmorrhage, it is interesting to consider the factors that may avert death. Shennan (1934) found that death was due to external rupture in 195 (95 per cent) of 206 dissecting aneurysms that did not survive for more than a few days. If death is to be postponed it seems clear that some event must take place to make external rupture unlikely, and this can only be the relief of pressure in the aneurysmal sac. This happens when, as in Case 1, the aneurysm ruptures internally, so that the blood flow re-enters the original lumen of

the aorta. The effect of re-entry on the period of survival is shown in Table I, which is compiled from Shennan's (1934) figures.

TABLE I

EFFECT OF RE-ENTRY ON THE PERIOD OF SURVIVAL

	Number of cases	Survival for less than 5 weeks	Survival for more than 5 weeks
No re-entry	199	192 (96%)	7 (4%)
Re-entry	92	26 (28%)	66 (72%)

Re-entry is, therefore, almost a pre-requisite of survival, for if it does not take place 96 per cent of patients die within five weeks, whereas when the circulation is re-established in this way only 28 per cent die in the first five weeks. Table I also shows that if the patient survives for more than five weeks, re-entry has taken place in 90 per cent (66 of 73 cases). This observation has an important bearing on the diagnosis of chronic dissecting aneurysm. In acute dissections East (1939) has emphasized the almost pathognomonic changes in the circulation of the legs due to obstruction of the aorta, which often afford convincing clinical evidence of the diagnosis. But when re-entry takes place the circulation is re-established in the legs; there will, therefore, be no evidence of aortic obstruction in 90 per cent of cases after the fifth week. Our Case 2 is an example of this, for the blood pressure in the legs was at least as high as that in the arms.

Shennan (1934) pointed out that re-entry into the original lumen is particularly liable to occur when the dissection reaches some obstruction, since this will increase the pressure in the aneurysmal sac. Mayr (1925) claimed that re-entrance usually took place into the iliac arteries, where the division of the aorta obstructs the progress of the dissection. The point of re-entrance was noted in 61 cases in Shennan's series; 43 aneurysms re-entered the pelvic branches of the aorta. Our Case 1 is an example of an unusual site of re-entry, for the extension of the aneurysm was prevented by the diaphragm and re-entry occurred at that level.

It was suggested by Bostrom (1888) that re-entry always prevented external rupture. This claim is not confirmed by our Case 1, and was criticized by Shennan for of 92 cases in his series in which re-entry occurred, external rupture also took place in 39 (42 per cent). On the other hand, when re-entry did not occur, external rupture took place in 95 per cent. Thus, although re-entry is no guarantee against external rupture, it does afford a considerable measure of protection against that fatal complication. Indeed, if re-entry takes place and the patient survives for more than a few weeks, there is a good chance that the aneurysm will not subsequently rupture. This is shown in Table II by an analysis of the causes of death in chronic dissecting aneurysms based on Shennan's (1934) figures.

TABLE II

CAUSES OF DEATH IN 79 CHRONIC DISSECTING ANEURYSMS

Heart failure	34
Hæmorrhage from aneurysm	16
Chronic nephritis	8
Cerebral hæmorrhage	6
Miscellaneous	11
Obscure or unknown	4
Total	79

In our Case 2 there is, therefore, no reason to believe that the aneurysm must ultimately rupture, and survival has been recorded for more than 30 years (Graham, 1886).

Summary

Two cases of dissecting aneurysm, diagnosed during life and surviving in one case for three years and in the other for at least eight years, are described. The importance of re-entry in averting fatal rupture of the aneurysm is discussed, and its significance in the diagnosis of chronic dissecting aneurysm is indicated.

We are indebted to Professor Crighton Bramwell and to Professor S. L. Baker for their interest and advice and to Dr. E. Duff Gray for the radiograms. Professor Bramwell has kindly given permission to publish details of these cases, which were under his care.

References

Bostrom, E. (1888). *Dtsch. Arch. klin. Med.*, **42**, 17.
East, T. (1939). *Lancet*, **2**, 1017.
Graham, J. E. (1886). *Amer. J. med. Sci.*, **91**, 155.
Mayr, L. (1925). *Zbl. Herz. Gefasskr.*, **17**, 263.
Rottino, A. (1939). *Arch. Path.*, **28**, 377.
Sailer, S. (1942). *Ibid.*, **33**, 704.
Shennan, T. (1934). *Spec. Rep. Ser., Med. Res. Counc., Lond.*, No. 193.
Weiss, S., Kinney, T., and Maher, M. (1940). *Amer. J. med. Sci.*, **200**, 192.

DISSECTING ANEURYSM WITH SURVIVAL FOR THREE MONTHS AFTER RUPTURE INTO THE PLEURA

BY

MAURICE CAMPBELL

From Guy's Hospital

Received June 12, 1946

The following case of dissecting aneurysm is recorded because the patient survived for three months after a considerable amount of blood had ruptured into the pleural cavity.

It is hard to write of any aspect of dissecting aneurysm without making use of Shennan's (1934) monumental review of 300 cases. In recent dissecting aneurysms external rupture is much the most common cause of death. Where the intrapericardial aorta was involved (177 cases) rupture took place into the pericardium in 152, into the pleura in 6, and into the mediastinum in 5 cases. Where the aneurysm was completely extrapericardial (41 cases) rupture into the pleural cavity was relatively more common—into the left pleura 13 cases, into the mediastinum and pleura (generally left) 11, into the mediastinum 2, and into the lung 1 case. In the old "healed" dissecting aneurysms (79 cases) heart failure was the commonest cause of death (34 cases) but rupture into one of the cavities occurred 16 times and in some of these it was into the pleura.

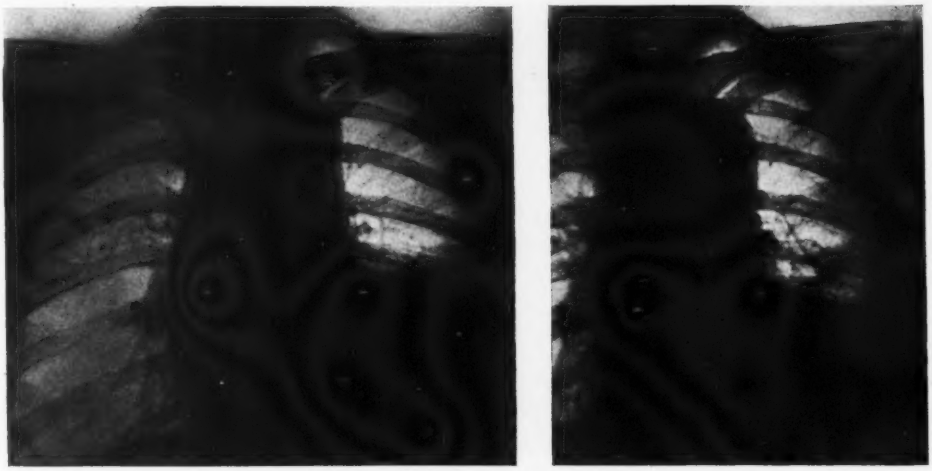
The following cases are the only references I have found in Shennan's monograph to hæmorrhage into the pleural cavities before the terminal episode, but his paper did not include a large number diagnosed during life. The case of Davy and Gates (1922) lived for 19 days and one of the reasons for the diagnosis was leakage of blood into the left pleura. The case of Barton (1930) lived for 17 days and fluid began to collect in the left pleura during the first day.

A woman was admitted to Guy's Hospital having collapsed after very severe pain in the chest and back with signs of fluid in the left chest. For ten years she had had occasional faints and for five years some pain on the left side of the chest, not clearly related to exercise. In September 1944 she had a right-sided hemiplegia from which she made a good recovery in a month; in December she had a similar attack, this time on the left side, and again recovered in the course of two months except for some slight residual signs.

On April 10, 1945, when 66, she was sitting on London Bridge Station waiting for a train when she was seized with agonizing pain in the centre of the chest and back. This was followed by breathlessness and collapse and she did not remember much about her admission to hospital. The blood pressure had fallen to 120/100 but soon rose to 220/130 and remained about this level after the first two days. The severest pain quickly subsided after morphia ($^1/_4$ grain and the same amount repeated) but she continued to have pain, especially in the back and needed a small dose of dilaudid ($^1/_{16}$ grain) most nights for some weeks. Her hæmoglobin had fallen to 55 per cent and there was a leucocytosis of 15,000 with a normal differential count.

Cardiac infarction was suspected but the cardiogram only showed slight inversion of T I and there was no significant change in this a week later. She was found to have a left-sided pleural effusion (Fig. 1A) and a week later aspiration showed almost pure blood, not merely a blood-stained effusion.

At this stage I was asked to see her and on these findings (with a loss of 3 stone during the previous three years) thought she must have a growth of the lung, possibly with a secondary deposit causing cardiac infarction, although the past cerebral history favoured primary cardiovascular disease and without the pleural effusion would have been taken as supporting cardiac infarction in spite of the absence of cardiographic support.



A B

FIG. 1.—Teleradiograms of the chest showing the wide aorta and large heart hidden in a left-sided hæmothorax. (A) 20/4/45, ten days after admission. (B) 14/7/45, three months after admission.

Progress. She made slow but steady progress although there was no significant change in the physical signs in the left lung. Five radiograms of the chest showed no great difference. The " aortic " shadow was wide but this was accepted as part of her atherosclerosis. After her death re-examination showed that the width had increased slightly from 8·5 to 9·5 cm.

After two months in bed she was allowed up and after another month was looking forward to going home. On July 10 she went to the lavatory and while there called and said she felt faint: her pain was not very severe but she collapsed and was carried back to bed. She became unconscious within a few minutes and died in half an hour.

Post-mortem examination. This was carried out by Dr. Hopkins to whom I am indebted for the specimen illustrated but the weights and full details of the other viscera were lost.

The brain was not examined. The kidneys were rather small and granular. The heart showed moderate hypertrophy of the left ventricle. The aortic valves showed some atheroma and calcification. The ascending aorta and arch showed slight atheroma only.

In the arch of the aorta, about midway between the origin of the subclavian artery and the level of the pulmonary artery there was a recent tear of the aorta about 3·5 cm. long, with an escape of blood into the lung and pleural cavity. This recent tear and the large amount of recent clot made it difficult to be sure if there had been an original tear here with rupture through to the lung and pleural cavity and downward dissection, or if the original leakage into the lung at this point had come from the lower tear (see later) with retrograde dissection back to this level.

Below this tear for at least 15 cm. there was an extensive dissection with stratified layers of blood clot, partially organized and looking of some months duration (i.e. probably from the time of the collapse and hæmothorax three months before). For 4 cm. below the tear (A to B, Fig. 2) there was a wider recent blood clot with a narrow tongue projecting a further 2 cm. (B to C), both on the inner side of the laminated clot, showing that the dissection had increased just before her death.

9 cm. below the fatal tear there was an old transverse tear 1·5 cm. long. The dissection extended below this and the stratified clot at this point looked at least as old as, and probably older than, anywhere else. This was certainly an old tear but it was not certain if it was the point of re-entry (though not the lowest point of the dissection) or, as seemed more likely, if it was the original tear causing the dissection that had spread upwards (as well as downwards) and had ruptured into the lungs and pleura near the point where the final tear had caused death. If this was so, it might explain why the second rupture into the pleural cavity had

FIG. 2.—Photograph of the upper portion of the descending aorta between the two tears. The original channel of the aorta has remained patent and the dissection is completely occluded with blood clot. On the inner side there is recent blood clot (from A to B) with a narrow tongue projecting down to C. On the outer side the blood clot is laminated and organized and near the lower tear the laminæ can be seen lying more horizontally and above this nore vertically. Above the upper tear part of the arch of the aorta has been cut away to show the blood clot penetrating into the lung.

caused death so quickly while the first had not. This uncertainty makes the case of little value except as an example of unusually long survival after a large hæmothorax had developed from a dissecting aneurysm.

SUMMARY

A case of dissecting aneurysm is described where the patient lived for three months with a large hæmothorax from the original tear and dissection of the aorta. She died suddenly from a second tear (also with some dissection) with rupture into the pleural cavity.

REFERENCE

Shennan, T. (1934). *Dissecting Aneurysms*, Medical Research Council Special Report Series, No. 193, London.

DISSECTING ANEURYSM OF THE AORTA: A NEW SIGN

BY

J. A. NISSIM

From the North Staffordshire Royal Infirmary

Received September 2, 1946

Ever since Shennan's (1934) masterly and comprehensive analysis of 317 cases of dissecting aneurysm of the aorta, including 17 of his own, interest in this condition has been steadily increasing, with the encouraging result of a mounting proportion of ante-mortem diagnosis. Shennan reported only 6 such correct diagnoses out of the 317 cases, up to and including 1932, and credited Swaine (1855–6) with being the first to make it. Reich (1944) reported a further total of 153 American cases since Shennan, 36 of which were diagnosed: he added 19 cases of his own but omitted Logue's (1943) remarkable series of 12 cases, of which 10 were correctly diagnosed. Thomas (1945) reported another case, also diagnosed during life. This alone brings the total up to 49 correct diagnoses out of 512 collected cases. The incidence of this disease as given by Shennan is one in every 175 autopsies. Reich in 1944 worked out the average incidence given by various writers as one in every 380 autopsies.

The effect of the stimulus Shennan provided has been kept alive by subsequent writings. This has resulted in a general change of attitude, to use the words of Glendy *et al.* (1937), "from one of remote academic interest in the pathological aspects of the subject to one of lively practical attention to the possibility of making an ante-mortem diagnosis." Apart from the description of new signs, the features that should provide a lead to the diagnosis have been amplified. The following case is recorded on account of its many interesting features. Diagnosis during life was expressed in these words: "Theoretically, the only diagnosis that would fit all the symptoms and signs of the patient is dissecting aneurysm of the aorta." In view of a suspicious peptic ulcer history during the previous year, however, and the presence of abdominal signs with release tenderness etc., an abdominal emergency, superimposed on a background of cardiovascular disease, could not be ruled out. Further, a sign, not hitherto mentioned in published writings, which promises to be of pathognomonic significance when present, will be described.

Case Report

A man of 71 years of age was sent into hospital with a short note from his doctor stating that the patient had a sudden attack of severe pain in his stomach the same day, and was thought to have a perforation of the stomach: $^1/_4$ of a grain of morphine sulphate had been given.

Seen by the resident surgical officer, the diagnosis of perforated peptic ulcer did not seem conclusive. Interrogation revealed that the patient was reclining in an armchair that morning, when suddenly he felt an extremely sharp and severe pain, a little below the umbilicus, soon spreading to the epigastrium and lower part of the chest, round both sides to the back, but not elsewhere. There was no retrosternal pain, and no nausea or vomiting. Slight dyspnœa was complained of. There was no disturbance of bowel movements, which had been regular up to and including that morning. For the past year he had some pain suggestive of peptic ulcer, which sometimes woke him up in the night and was often relieved by taking food. He also gave a history of high blood pressure for some time, and typical anginal pain, which he had experienced on four or five occasions; the pains came on with exertion and disappeared fairly quickly with rest.

On examination he showed an anxious pale face, and was obviously in severe pain, but could move about in bed. His temperature was subnormal and his respiration about 20 a minute. The abdomen was found to move well with respiration, but there was tenderness and guarding all over. Release tenderness was elicited as well, especially from the epigastric and right hypochondriac regions. There was no shifting dullness, no palpable abdominal mass, and no abnormal sounds on auscultation.

The heart was not obviously enlarged. A radiogram of the chest, however, taken the next day about two hours before death, showed a rather enlarged heart shadow with a widened supracardiac shadow. The heart sounds were faint but regular. There was persistent bradycardia of 44 beats a minute, but the previous rate was unknown. No murmurs or other adventitious sounds could be heard. Examination of the lungs, the central nervous system, and the urine yielded no abnormal findings.

Some condition simulating coronary thrombosis that could explain these signs and symptoms was sought and dissecting aneurysm of the aorta seemed a possibility. The radial pulses on the two sides were found different in strength and confirmation was obtained by the sphygmomanometric reading of of 80/? on the right and 120/? on the left. The dorsalis pedis and posterior tibial arteries were easily felt in both legs and found of equal volume. A slight transient " peculiar " feeling in his head was later mentioned by the patient to have preceded the onset of the severe pain in the infra-umbilical region by a short while.

Another finding that came to notice later was a difference between the right and left carotid pulsations. They were reduplicated on the right and single on the left, without a coexisting noticeable difference in the intensity of the pulsations on the two sides. There were two almost equal beats in quick succession on the right carotid for every single beat on the left. The right radial pulse, however, like the left, was single but weaker. Electrocardiography could not be carried out owing to an unfortunate breakdown of the apparatus.

The same evening, the pulse rate dropped to 36, although his general condition was improved. The next day it went up to about 55 a minute. Towards the evening the patient developed an acute exacerbation of the symptoms and of the abdominal signs—excruciating epigastric pain radiating below the umbilicus and upwards into the lower chest, with guarding that almost amounted to rigidity. About an hour later he gave a few gasping breaths and died suddenly.

Necropsy (Dr. A. J. McCall). The body was that of a rather emaciated man.

Skull and brain. These were not examined.

Thorax. There was a hæmopericardium with 600 g. of recent blood clot closely investing the heart. The aorta showed a 2·5 cm. horizontal tear about 4 cm. above the aortic ring. At right angles to this and running into it was a longitudinal tear, which had curled round beneath the adventitia. In one place, the adventitia had given way, giving rise to the hæmopericardium. Blood had spread beneath the epicardium and into the auricular septum where hæmorrhages were present in close relation to the A-V bundle. Distally, the blood had dissected the aorta down to the fourth lumbar vertebra, where a second tear allowed re-entry into the aorta. Dissection had occurred along the innominate artery, but not along the left subclavian or left carotid arteries, so that the difference in blood pressure during life on the two sides was explained. The aorta at the site of the rupture was thinned but not otherwise obviously abnormal. Distally, however, there was much atheroma and some pearly intimal thickening.

Heart. This was not unduly large. The coronary arteries showed only slight atheroma.

Abdomen. Some blood had effused behind the peritoneum in front of the pancreas and in its substance. The spleen was slightly enlarged and firm. The abdominal organs were otherwise healthy.

Discussion

Clearly the terminal event causing the sudden death was the rupture into the pericardial sac, with a consequent hæmopericardium and tamponade. The partial heart block could be explained by the blood seeping into the auricular septum and across the A-V bundle, and the low abdominal pain by extension of the dissection as far down as the fourth lumbar vertebra, but why the pain was first manifest there was not clear. The severe epigastric pain simulating an acute abdominal emergency seems to be best explained by the hæmorrhage in front of and in the substance of the pancreas.

The only single diagnosis to explain the multiplicity of signs and symptoms seemed that of dissecting aneurysm of the aorta. In the absence of pathognomonic signs, however, the suspicious past history of the patient with a double retrospective diagnosis of peptic ulcer and angina pectoris, and the obvious abdominal signs including release tenderness, especially for the first few hours, combined to make the consideration of other conditions in the differential diagnosis seem necessary.

Cardiac infarction seemed unlikely and syphilitic aneurysm of the upper aorta could cause the pain in the lower chest and the difference in the brachial blood pressure, but other corroborative signs were missing. Acute abdominal conditions could not explain the brachial difference in blood pressure and the partial heart block, but it was thought that they might

supervene in a case that had those signs already from preceding hypertension, arteriosclerosis, and myocardial degeneration. Not long before, a patient suspected of having aortic aneurysm because of a difference in brachial blood pressure of 30 mm. was shown at necropsy to have bronchial carcinoma and a deformed right clavicle.

Even after dismissing the cardiac signs as coincidental, any of the abdominal conditions thought of seemed to leave a great deal unexplained. Mesenteric embolism lacked its usual abdominal distension, colicky obstructive type of pain, and nausea and vomiting. Acute pancreatitis was more seriously considered, but the onset seemed rather too sudden and still did not explain the initial low pain. Perforation of a peptic ulcer lacked its board-like rigidity and restriction of abdominal mobility with respiration. The subsidence of the abdominal pain without the supervention of a peritonitic stage at the time of re-examination by the surgeon seemed almost to rule it out.

It is interesting to note what Shennan wrote regarding the course of the pain. " In some cases a very characteristic feature which gives an important clue to the condition present is the gradual disappearance of the pain, the relief persists for hours or days or even a week or two, but the pain recurs later with similar rapidity and rapid death." Although the relief in this case was far from complete, the sequence of events seems to be very similar.

The clinical manifestations of dissecting aneurysm of the aorta are protean. Simple meditation on the anatomical aspects would soon conjure up an almost endless chain of symptoms, signs, and differential diagnoses. It is the coexistence of certain groups of signs and symptoms rather than any single one of them that should almost always lead one to suspect the condition. Sometimes, however, it is a long way from making certain of the diagnosis.

Among the leading symptoms of this catastrophe, the pain with its type, mode of onset, and distribution is of paramount importance in diagnosis. It is usually very severe, extremely sudden, and is described as splitting, tearing, rending, crushing, or knife-like in character. As a rule, it is felt in the midthorax, front or back, and usually descending from above downwards. It may be felt in the neck, mandible, head, beneath the left or right scapula, in the shoulders, lumbar regions, loins, epigastrium, right or left hypochondrium, right or left iliac fossa, and infra-umbilical region. It may also radiate into the legs, as far down as the toes, but radiation into the arms is unusual. Typical coronary thrombosis pain has also been described from extension of the dissection into the ostia of the coronary arteries. Sweating, faintness, shock, and collapse are frequent and may be extreme. Palpitation from extrasystoles or ventricular tachycardia may be complained of. Dyspnœa is frequently reported and appears to be more pronounced than is generally realized; it may be severe and lead to orthopnœa. Another important group of symptoms is due to the dissection extending along the peripheral vessels: along the external iliacs producing numbness, coldness, and weakness of the legs; along the coeliac axis or mesenteric vessels producing nausea and vomiting, diarrhœa, hæmatemesis, melæna, abdominal cramps, or distension; or along the renal vessels, producing hæmaturia and anuria. Even subcutaneous hæmatomata have been recorded, from dissection of the superficial vessels. Involvement of the cerebral arteries can result in a whole gamut of symptoms from headache to coma: visual disturbances, facial paralysis, loss of speech, hemiplegia, paraplegia, visceral pains of tabetic type, and incontinence. A third variegated group may be met with from rupture of the dissecting aneurysm anywhere inside the chest, and from pressure effects so produced: cough, sensation of suffocation, dysphagia, hoarseness, and hæmoptysis. Symptomless cases are known to occur.

A sign has been met with in the present case to which no reference could be found. This was the unilateral reduplicated beat of the carotid artery. The explanation must lie in the difference of the rate of propagation of the pulse wave through the lumen of the artery and through its dissected coats where the blood was probably partly clotted. At necropsy, this was the only pathological finding that could account for it. The full significance of this sign was not realized until then. White (1944) in his authoritative summary of dissecting aneurysms says, " There are no pathognomonic signs of dissecting aneurysms." Since, however, there does not seem to be any other pathology that could produce this unilateral double beat in an artery, its pathognomonic significance is at once appreciated. The frequency and usefulness of this sign may be greater than is at present realized, and it may occur in other arteries. Looking for this sign by palpation of all accessible arteries may reveal further instances.

Two other signs of dissecting aneurysm have been added in recent years. Roesler, Gifford, and Betts (1937) described the appearance of a rapidly shifting area of pulsation in the interscapular area, over which the aortic second sound was very accentuated: it was also associated with a rapid change in the radiological appearance of the aortic shadow. The other sign was due to Logue (1943) and consisted of a bruit and a thrill over the femoral artery.

Delay in the conduction of pulse beats is also significant. " In dissecting aneurysm of the aorta delay of the pulse beat, when compared with the apical beat, can occur in all arteries, resulting from delay and weakening of the blood stream entering or passing through the involved artery or arteries " (Shennan, 1934). Appreciable help may be obtained from the search for these simple signs in suspected cases.

Summary

A review of the improved diagnosis of dissecting aneurysm of the aorta during life is presented. Its manifestations are protean, but some leading symptoms are singled out.

A case in which this diagnosis was considered as the only one to include all the signs and symptoms under a single pathology is described.

A new sign of this disease is described consisting of unilateral reduplicated arterial pulsation, in this case in the right carotid artery. Its pathognomonic significance is discussed, and other recently described signs are reviewed.

I should like to render my thanks to Dr. A. Wilson Gill for his help and Dr. A. J. McCall for the use o the post-mortem report.

References

Glendy, R. E., Castleman, B., and White, P. D. (1937). *Amer. Heart J.*, **13**, 129.
Logue, R. B. (1943). *Amer. J. med. Sci.*, **206**, 54.
Reich, N. E. (1944). *Clinics*, **3**, 346.
Roesler, H., Gifford, U. G., and Betts, W. (1937). *Amer. Heart J.*, **13**, 426.
Shennan, T. (1934). *Dissecting Aneurysms*, M.R.C. Report No. 193, H.M. Stationery Office, London.
Swaine (1856). *Trans. Path. Soc.*, **7**, 106.
Thomas, G. T. (1945). *Clinical J.*, **74**, 20.
White, P. D. (1944). " Heart Disease, ' 3rd ed., Macmillan, New York.

ANEURYSMAL DILATATION OF THE LEFT AURICLE WITH EROSION OF THE SPINE

BY

H. ASHWORTH AND A. MORGAN JONES

From the Cardiographic Department, Manchester Royal Infirmary

Received May 19, 1946

Although aneurysmal dilatation of the left auricle, due to mitral stenosis, is not uncommon, the following case is unusual for the enlarged auricle eroded the vertebral column, causing severe pain in the right chest, and became adherent to the right chest wall, leading to systolic retraction in the right axilla.

Case Record

In August, 1945, a single woman, aged 38, employed in clerical work, was admitted to the Manchester Royal Infirmary, under the care of Dr. Crighton Bramwell, on account of severe pain in the right chest of four years' duration. Apart from scarlet fever and an antral operation in childhood, her health had been good until the age of 22 when she developed rheumatic fever and was treated in hospital for five months. She was subsequently troubled by occasional faintness and palpitation but carried on her occupation as a clerk, with little time off work owing to ill-health, until the age of 26 when she had an attack of lobar pneumonia. She never again resumed her occupation and became unable to undertake household duties owing

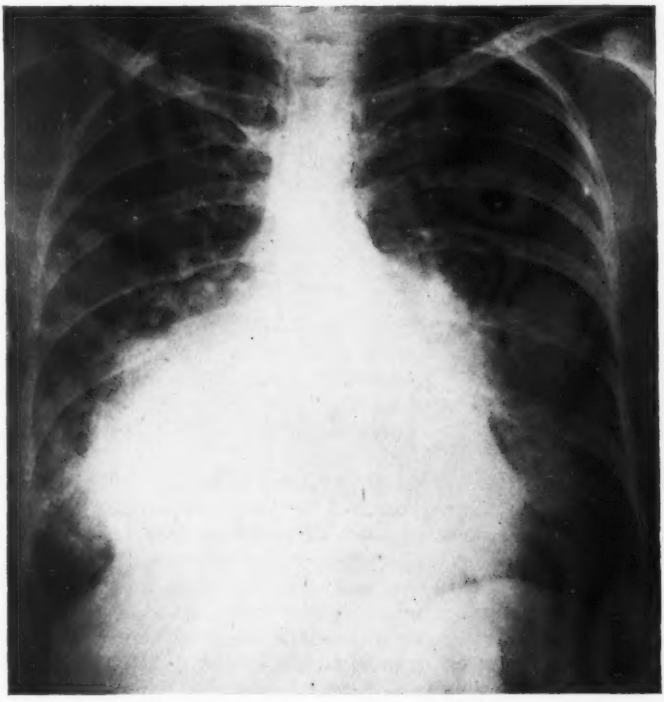


Fig. 1.—Teleradiogram, 1943. Anterior view, showing great dilatation of left auricle to the right.

to severe dyspnoea on exertion. Aged 34, she began to suffer from gripping pain starting at the inferior angle of the right scapula and radiating round the chest to the right axilla. This occurred on exertion, on swallowing, or when her stomach was distended with food, and was relieved by rest and sometimes by alkalis. In July 1943, when aged 36, she was seen by Dr. Bramwell on account of this pain, and was found to be suffering from severe mitral stenosis with gross cardiac enlargement and uncontrolled auricular fibrillation. On cardioscopy, the left auricle was grossly dilated (Fig. 1 and 2). She was admitted to the Manchester Royal Infirmary,

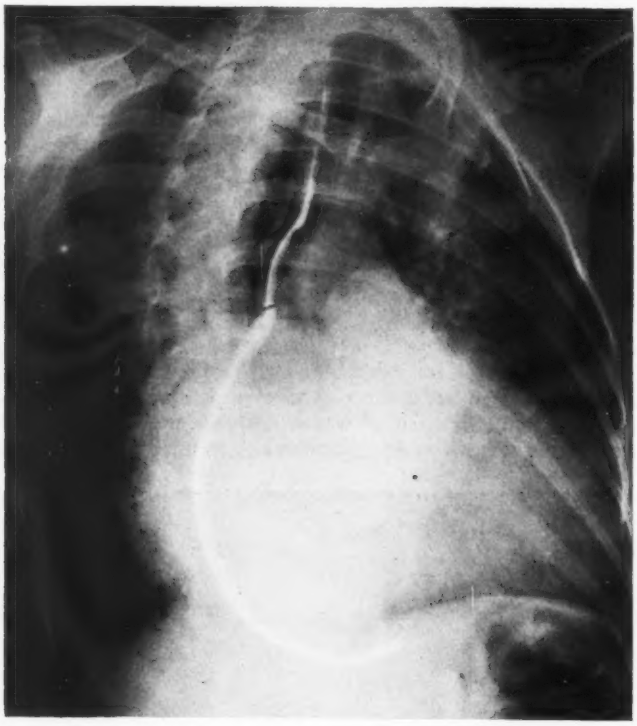


FIG. 2.—Right anterior (I) oblique radiogram, 1943, showing displacement of barium filled oesophagus by left auricle.

under Dr. Bramwell, and was given digitalis with improvement in her capacity for exertion. She remained under our observation, with persistent fibrillation, and, apart from the pain, relatively well, until August, 1945, when she was readmitted to hospital because her pain, hitherto tolerable, had become very severe and almost continuous, being no longer relieved by rest. A cardiogram six days before admission showed auricular fibrillation with right axis deviation (Fig. 3A).

On examination she was much wasted, and distressed by pain. The pulse was completely irregular, rate 136 a minute; blood pressure 130/90 mm. The heart was enlarged; at the apex a very loud first sound and a long rumbling diastolic murmur were heard. There was no evidence of right ventricular failure. The right lower chest was dull on percussion and breath sounds and tactile fremitus were almost absent; moist sounds were present at both lung bases. Systolic retraction was conspicuous in the sixth, seventh, and eighth intercostal spaces in the right mid-axillary line. On cardioscopy the heart was grossly enlarged and the right lower chest was radio-opaque. Right oblique and lateral views failed to show any erosion of the spine; unfortunately left oblique views were not taken.

Two days after admission normal rhythm with a prolonged P–R interval was recorded (Fig. 3B) and, apart from several attacks of regular tachycardia (Fig. 3C) lasting some hours,

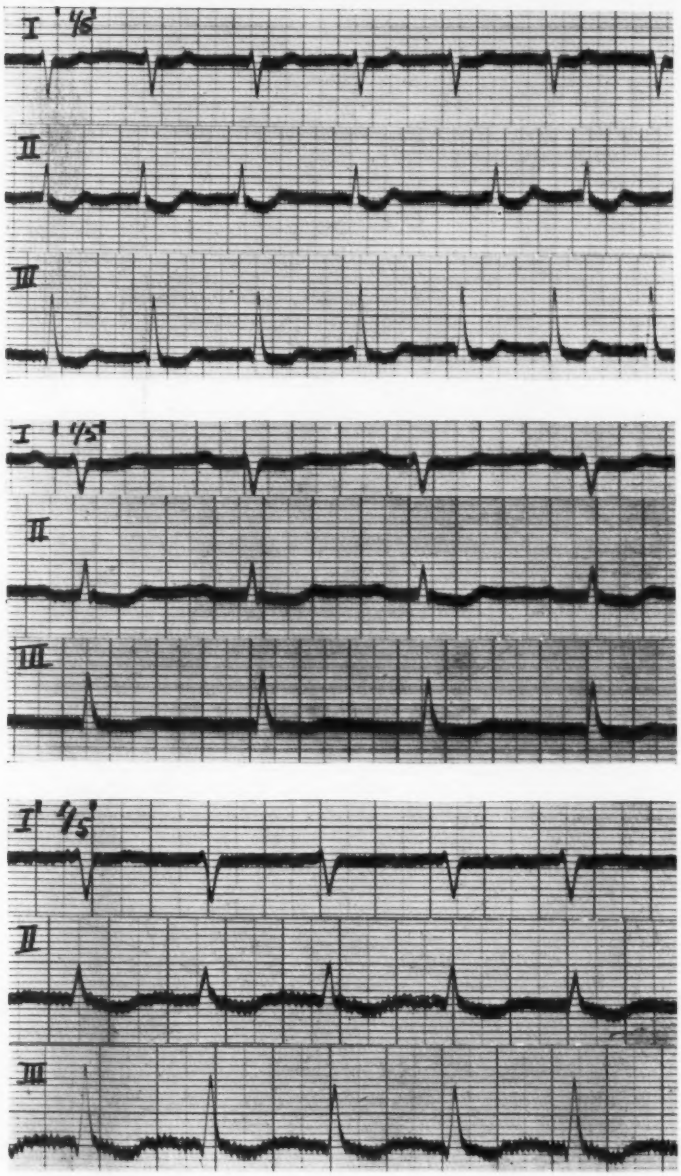


FIG. 3.—(A) Cardiogram six days before admission, showing auricular fibrillation with right axis deviation and digitalis effect.
(B) Cardiogram two days after admission, showing sinus rhythm with a prolonged P–R interval.
(C) Cardiogram during an attack of regular tachycardia, rate 140 a minute. (Calibration: $3mV$= 2 cm.)

this persisted until her death seven weeks later. In spite of treatment her condition gradually deteriorated; she became more feeble and wasted, and her pain increased in severity, being relieved only for short periods by full doses of morphia. Two days before death slight oedema of the legs appeared, but there was no gross right ventricular failure.

Necropsy was performed by Dr. F. A. Langley, 23 hours after death. The heart weighed

FIG. 4.—Section of left auricular wall stained with haemalum and eosin. Although there is some fibrosis, a considerable number of muscle fibre bundles can be seen. Magnification: ×36.

A B

FIG. 5.—Post-mortem radiograms of the dorsal spine. (A) Lateral view which fails to show the erosion. (B) Left anterior oblique view showing erosion of the right antero-lateral aspects of the vertebral bodies. The deeply eroded vertebra is the seventh dorsal.

420 g. The right border, formed by the left auricle, extended 10 cm. to the right of the midline. The lower lobe of the right lung was partially collapsed and firmly adherent to the chest wall laterally and to the pericardium and left auricle medially. The left auricle was enormously dilated and its wall thin; microscopy of the wall revealed some fibrosis, but a considerable amount of muscle tissue was present (Fig. 4). The right auricle was moderately dilated; the right ventricle was 3–4 mm. in thickness. The left ventricle was moderately dilated, the wall measuring 1 cm. in thickness. The mitral valve was severely stenosed and reduced to a narrow slit 1·5 cm. in length; the tricuspid valve was slightly stenosed (circumference 10·5 cm.) and its cusps and chordæ tendinæ were thickened. The aortic cusps were slightly thickened; the pulmonary valve appeared healthy. The fifth, sixth, seventh, eighth, and ninth thoracic vertebral bodies were eroded, the intervertebral discs being spared. The erosion of the seventh vertebra was most conspicuous (Fig. 5). Apart from some chronic venous congestion, the remaining organs were healthy.

Discussion

Erosion of the spine by a dilated pulsating auricle lying against it does not seem surprising, but we have been unable to find any report of a similar case. Since importance has been attached to pulsation as a factor in the mechanism of erosion of bone by arterial aneurysms, it is interesting that a fibrillating auricle should be capable of similar erosion. It is tempting to suggest that reversion of the auricle to normal rhythm played a part in the exacerbation of the pain in the later stages. The presence of vertebral erosion combined with the distribution of pain strongly suggested root pressure, which would explain the intractable character of the pain. Had this possibility occurred to us during life, paravertebral root injection might have been tried, for there seems little doubt that the severe continuous pain played an important part in the progressive deterioration of the patient's condition that ultimately led to death. In the earlier stages pain was provoked by exertion and relieved by rest. It is possible that the rise of venous pressure during exertion, by further distending the auricle, increased root pressure, and that the fall in auricular pressure with rest reduced it. Pain in the right chest on exertion in a case of aneurysmal dilatation of the left auricle was described by Bedford (1927) but necropsy findings were not available. Systolic pulsation on the right side of the chest has been recorded by Dressler (1937) and by Bedford (1927) but we have not found a case in which systolic retraction was noted.

It seems surprising that, after years of fibrillation, the enormously dilated left auricle should have been capable of reverting spontaneously to normal rhythm. We have, however, seen normal rhythm return in a case of greatly dilated left auricle in which fibrillation had persisted for 19 years; but in that case fibrillation recurred within a week. The amount of muscle tissue in the auricular wall in the present case was much greater than that usually found in an extremely dilated left auricle and this may have facilitated reversion to normal rhythm.

Summary

A case of aneurysmal dilatation of the left auricle associated with mitral stenosis is described in which severe pain in the chest was a prominent symptom. Necropsy showed erosion of the bodies of several dorsal vertebrae, due to pressure from the dilated auricle. Systolic retraction of the chest wall in the right axilla and spontaneous reversion to normal rhythm after fibrillation had persisted for several years were other unusual features of the case.

We are indebted to Professor Crighton Bramwell for permission to publish details of this case, to Dr. E. Duff Gray for the clinical radiograms, to Dr. F. A. Langley for his help with the pathology, and to Mr. F. Ward for the post-mortem radiograms and the microphotograph.

References

Bedford, D. E. (1927). *Amer. Heart J.*, **2**, 127.
Dressler, W. (1937). *Arch. intern. Med.*, **60**, 663.

EARLY DIAGNOSIS OF RHEUMATIC VALVULAR DISEASE IN RECRUITS

BY

JOHN PARKINSON AND RONALD HARTLEY

From the Cardiac Department of the London Hospital

Received September 25, 1946

During the world war of 1939–45 the call for men for the armed forces made the medical examination of potential recruits an important field of medical service, and many were rejected for heart disease. The major problem for cardiologists was to detect the presence of rheumatic heart disease when it was early and symptomless, a stage not seen except incidentally in peace-time practice. It might at first be thought that with the advent of peace the importance of very early diagnosis would quickly fall. But it is becoming evident that in economic and labour conditions after this war, comparable medical examinations will be multiplied and that in time few employees will be accepted without them.

From 1940 to 1945, 2500 men suspected of cardiac abnormalities were referred to one of us (J. P.) by medical boards. Among them were found 609 cases of early rheumatic heart disease, of which only a quarter had symptoms, and those slight. Only 264 had mitral valvular disease, a low proportion due to the fact that mitral stenosis is more easily identified by medical boards than aortic incompetence, and that those with mitral disease more often get symptoms at an earlier age and, therefore, come under medical observation. It is on the analysis and study of these 609 together with a limited survey of the literature, that this paper is mainly based. Table I shows the relative incidence of various cardiovascular defects. Cases of effort syndrome (neuro-circulatory asthenia) were not included.

TABLE I

CARDIOVASCULAR DISEASE AMONG 2500 MEN REFERRED BY MEDICAL BOARDS

Rheumatic heart disease	609	(76 per cent)
Congenital heart disease	71	(9 per cent)
*Hypertension	49	(6 per cent)
Enlargement, cause unknown	25	(3 per cent)
*Hyperthyroidism	12	
Angina of effort	12	
Coronary thrombosis	5	
Syphilitic aortic incompetence	4	
Parox. tachycardia	10	
Bundle branch block	2	
Complete heart block	1	
Auricular fibrillation	5	
Auricular flutter	1	
	806	

* These figures are smaller than might be expected because many such cases were referred to physicians other than cardiologists.

The figures in Table I may be compared with those given in a table shown among the individual reports of the chairmen of the special medical advisory boards in the United States (Fenn *et al.*, 1944). Of 698 rejected for cardiovascular disease and disorder, 396 had rheumatic heart disease, 43 had congenital heart disease, and 161 had hypertension; neuro-circulatory asthenia accounted for 61 rejections.

The proportion of cardiovascular disease among recruits in this country has been given as

9 per cent of 30,000 rejections for all causes ; of this 9 per cent, four-fifths were for rheumatic heart disease (Parkinson, 1945). In the United States, Rowntree (1945) estimated that 6 per cent of 2,976,000 total rejections were for cardiovascular reasons. About half were due to rheumatic lesions (Levy, Stroud, and White, 1943). Wilburne and Ceccolini (1944) examined 20,000 men and found that 6 per cent of all rejections were for cardiovascular lesions and that 63 per cent of these were rheumatic. Perrot (1944) found that 8 per cent of all rejections were for cardiovascular reasons.

Method of Examination

Past History. No great reliance was placed upon a past history of rheumatic fever or chorea, whether negative or positive; too many men, in their eagerness to serve or to escape service, will falsify such a history; the same applies to a story of dyspnœa on exertion.

Inspection. This served to exclude obvious anæmia or goitre, and to note nutrition, physique, and any deformity of the chest or spine.

Palpation. The apex beat was then found by placing the whole hand lightly upon the chest wall under the left breast. It was noted whether the impulse was displaced or too forcible. Minor degrees of scoliosis frequently move the apex beat outwards. The anterior chest wall was then palpated for the presence of a thrill, the palm of the hand being laid lightly over the apex and then over the base of the heart. A thrill at the base of the heart may best be felt by the observer standing on the right of the subject who leans forward and stops breathing at the end of full expiration ; the hand should be merely applied to the chest wall, not pressed hard. No reliance whatever was placed upon a doubtful thrill at any site.

Auscultation. The heart sounds were then considered, whether normal or abnormal, their loudness, and any variations in character which frequently lead to doubt in diagnosis. A split first sound which is fairly common in health sometimes led to an unjustified suspicion that early mitral stenosis was present; an accompanying double quality in the feel of the apex beat added to this suspicion. The first sound may otherwise vary in loudness or clarity in different subjects without significance. It is often said that a pulmonary second sound, loud or duplicated or both, is a sign of early mitral disease. But this is not so—such variations are common in healthy young adults. The normal third heart sound, so common in the young (Thayer, 1909 ; Evans, 1942*b*), was also frequently mistaken for a mitral diastolic murmur. Bramwell (1943) found a reduplication of the second sound at the apex, which he considers to be identical with the third heart sound, to be frequently mistaken for mitral stenosis.

Murmurs were first considered with the subject standing, then when lying, and again when in the left lateral position after exercise. During this procedure a presystolic murmur is heard best at the higher range of rate, and an aortic diastolic during the lower range of rate. We have found it convenient thus to listen for an aortic diastolic murmur on the left of the sternum while the heart rate is falling after the subject has exercised and turned half over on his left side, and immediately after deciding on the presence or absence of a presystolic murmur at the apex. The aortic diastolic murmur is also well heard with the subject leaning forward with the breath held at the end of full expiration. We have often been exhorted to keep an *open* mind, and in general this is sound advice. We do naturally listen without preconceived ideas of what will be heard; and we cannot help noticing a first sound which is unusually loud or when there is an obvious systolic murmur. That is easy, and now comes the moment to *close* the mind to what is likely to preoccupy it and to concentrate upon answers to special questions :

(1) Is there a mid-diastolic murmur at the apex, a distant murmur beginning with the third heart sound but distinguishable from it ? If so there is mitral stenosis. (2) Is there a diastolic murmur immediately following the second sound? If so there is aortic incompetence and the murmur can be tracked up the left border of the sternum to its aortic source. Murmurs were timed by the carotid pulse, felt best with the observer's left hand resting on the right side of the patient's neck, the thumb on the artery.

The Pulse. The rate and rhythm of the pulse were noted at the end of the examination, when the recruit was more composed. Its response in rate to effort was noted when auscultating after exercise, but no so-called exercise tolerance tests were made.

Radioscopy. Radioscopy was used in every case, employing the three standard positions, i.e. anterior, right oblique, and left oblique ; and all statements on heart size were based on radioscopy, occasionally supported by teleradiography.

Electrocardiography. Cardiograms were taken on the first 500 cases but their value was so limited that subsequently they were taken only for extra evidence when diagnosis was difficult or when an unusual rhythm was present. This agrees with the conclusions of Wood, Wolferth, and Miller (1941) based on a large experience. The exigencies of war practice had also to be considered. But a cardiogram was always taken in suspected myocardial disease, chiefly among the older recruits.

The Diagnosis of Early Mitral Valvular Disease

This entails a discussion of the significance of the apical systolic murmur which has been regarded as the commonest murmur in mitral stenosis (Graham Steell, 1906). We doubt whether this is true of young adults judged by our experience with recruits. Of 184 recruits diagnosed certainly as mitral stenosis, 104 had a presystolic murmur (in 72 alone, in 3 with a diastolic, and in 29 with a systolic murmur in addition) ; 13 had a mitral diastolic murmur only; 5 a loud apical first sound only; and 2 were diagnosed solely on the X-ray appearances, there being no abnormal auscultatory sounds. A further 60 had only a systolic murmur. One might expect to find in current textbooks, under mitral stenosis and its diagnosis, a special reference to the first signs permitting its early diagnosis ; but the description is usually that of the fully developed disease as seen in patients with symptoms seeking medical advice. In contrast, war requires, and peace should require, the recognition of the symptomless valve lesion.

The Apical Systolic Murmur. The commonest cause for a medical board to want a cardiologist's opinion was the presence of an apical systolic murmur. If it was not overlooked, the common soft pulmonary systolic murmur was apparently more easily assessed as insignificant.

We have reserved the term incidental—preferring it to the term functional—for those systolic murmurs that occur not only in health and, therefore, could be classed as innocent, but also in anæmia and pyrexia—murmurs that are not indicative of cardiovascular disease. Systolic murmurs also occur with " relative incompetence " of the mitral valve, as in association with aortic incompetence or stenosis, in hypertension, rheumatic carditis, subacute bacterial endocarditis, and heart failure. Rheumatic heart disease affecting the mitral valve is a third and the most important source of this sign. A harsh systolic murmur with thrill near the sternum in the fourth or fifth left interspace may be congenital—that of a ventricular septal defect.

The incidence of systolic murmurs in any group of apparently healthy people is high. Contratto (1943) heard such murmurs in 350 (12 per cent) of 2856 college students examined ; of these, 208 were classed as functional, 9 as rheumatic valvular disease, 6 as congenital lesions and 127 were regarded as more than functional, though the physical signs were insufficient to permit an exact diagnosis. Wilburne and Ceccolini (1944) found 297 functional murmurs among 20,000 recruits. In our own series 259 (10 per cent) incidental murmurs of impressive intensity were heard.

Studies of the natural history of the systolic murmur such as those by Blumenthal (1942) and by Baker, Sprague, and White (1943) underline the great difference in prognosis between the incidental (or functional) murmur and the organic mitral systolic murmur. The difficulties in the assessment of the systolic murmur are considerable; Master (1944) asserted that many men admitted to the U.S. Navy with so-called functional murmurs were later boarded out with valve lesions.

Incidental murmurs are generally soft and short, and they vary in intensity both with position and with respiration to a greater degree than organic murmurs. They are unaccompanied by symptoms or signs of heart disease. Radioscopy shows a heart normal in shape and size. There may be some chest abnormality such as a depressed sternum which is often associated with a systolic murmur, an association noted twelve times among our 2500 recruits. The term " cardio-respiratory " applies by definition to systolic murmurs varying with respiration, disappearing at some phase, often that of full expiration or inspiration. In practice

they are short and soft, and seldom present difficulty. Exocardial murmurs were heard in three men. Scadding and Wood (1939) have drawn attention to a systolic click that occurs in cases of left-sided early pneumothorax.

The important features in assessing a systolic murmur are loudness and length, the presence or absence of a diastolic murmur, and evidence of enlargement of the heart on X-ray. As regards the intensity, White (1927) showed that there is a distinct difference in prognosis between the three groups of loud, moderate, and slight murmurs. Levine (1933) and Freeman and Levine (1933) advised that systolic murmurs be graded into six groups according to their intensity: they found that only the first two groups of this classification were of no significance and that the majority of functional murmurs fell into the first group, which consisted of the very shortest and softest murmurs.

Evans (1942*a*) has lately again stressed the importance of the late systolic murmur, i.e. a murmur occurring late in systole, separated from the first heart sound by a short interval; although this murmur is often noticeable, Evans believes that it is always innocent. Twenty-four such murmurs were heard among the 2500 recruits seen, and there was never any reason to think they represented mitral disease. A medical board, however, might regard them as evidence of mitral stenosis or of aortic valvular disease (Table II).

TABLE II

ANALYSIS OF THE DESCRIPTION OF 24 "LATE SYSTOLIC MURMURS" AS STATED BY MEDICAL BOARDS WHEN SEEKING A CARDIOLOGIST'S OPINION

(1) Systolic murmur at the apex	7
(2) No reference to the murmur	3
(3) Apical murmur with no reference to timing	4
(4) Diastolic murmur	4
(5) Presystolic murmur	2
(6) Systolic and diastolic murmurs	2
(7) Board stated difficulty in timing the murmur	2
	24

The intensity of murmurs may be graded simply into three groups, soft, moderate, and loud, and the length may be given as short, moderate, or long. The phrase "the murmur replaces the first sound" used often to be employed, but now it is assumed that if the systolic murmur is long and loud enough the first sound may be ousted. The conduction of a murmur is little more than a measure of its intensity (Levine and Likoff, 1944). We join with Levine (1933) and Freeman and Levine (1933) and Bramwell (1942) in hesitating to call a loud systolic murmur incidental or functional, even in the absence of collateral signs of heart disease. It is in such cases that the routine use of the phonocardiograph may be of great use in the near future. Whether a recruit should be accepted for the forces when he possesses a very noticeable but innocent systolic murmur is open to argument. We think that unless extremely keen, such a man runs the risk of a "doctor made" cardiac neurosis (Hill and Dewar, 1945). Some claim that this is placing the individual before the State, but such a soldier may become more of a liability than an asset in any active unit.

MITRAL STENOSIS WITH A PRESYSTOLIC MURMUR

One hundred and four (almost 40 per cent of all the cases of mitral valvular disease) cases of mitral stenosis with a presystolic murmur, and all with a loud first sound, were seen. Of these 37 per cent gave a history of rheumatic fever, 15 per cent of chorea, and 3 per cent of both (total 55 per cent). Among men without heart disease a history of rheumatic fever was given in 14 per cent, of chorea in 2 per cent, and of both in 0·1 per cent, a total of 16 per cent. This figure is higher than in the normal population because such histories often influenced the medical boards in deciding to seek a cardiologist's opinion. More reliable percentage frequency of rheumatic history in the normal is that of 1000 men studied during the first world war, namely 5 per cent (Parkinson, 1945).

Dyspnœa on exertion was the only symptom considered; in all cases of mitral stenosis, no matter what the physical signs, the proportion with dyspnœa proved to be about the same, 20 per cent. Short-windedness must always be considered in relation to the build, occupation,

and condition of the patient. Many men with mitral stenosis showed a loud and sudden first sound as the only obvious physical sign. In the majority, however, a presystolic murmur was evoked by exertion and then heard in the left lateral position. In the absence of a presystolic murmur, and in the absence of tachycardia, the characteristic apical first sound points to the probability, but not the certainty of mitral stenosis. Other causes of a loud first sound are hypertension, tachycardia, and thyrotoxicosis, and in the last especially it can resemble a presystolic murmur. In five cases with a loud and sudden first sound only, and tachycardia absent, exertion failed to elicit a presystolic murmur; yet the X-ray appearance was, in all five, diagnostic of mitral stenosis. Thirteen cases had a mitral diastolic murmur only, limited to the apex. The percentage having symptoms was essentially the same in this last group as in those with a presystolic murmur; the percentage with a positive rheumatic history was also identical with that of the presystolic group.

TABLE III

AUSCULTATORY SIGNS IN 184 CASES OF EARLY MITRAL STENOSIS

Presystolic murmur only	72
Presystolic murmur and systolic murmur	29
Presystolic and diastolic murmur	3
Loud mitral first sound only	5
Diastolic murmur only	13
Systolic murmur only	60
No abnormal auscultatory signs	2
	184

MITRAL STENOSIS WITH A MURMUR, SYSTOLIC NOT PRESYSTOLIC

In a total of 264 cases seen with mitral valvular lesions, 60 had a mitral systolic murmur of moderate or loud intensity and of moderate or long duration. This with a combination of an apical systolic thrill in 9 ; a rheumatic history in 48 per cent, chorea in 7 per cent, and both in 5 per cent (total 60 per cent) ; dyspnœa on exertion ; and a loud first sound, or X-ray changes (90 per cent), allowed a diagnosis of mitral stenosis to be made with some certainty.

MITRAL INCOMPETENCE

Between the two world wars it was frequently taught that mitral incompetence was rare, and incidental (or functional) murmurs were diagnosed freely. A mitral lesion without stenosis is not commonly seen at necropsy. Cabot (1926) found only seven cases of mitral incompetence in 4000 autopsies, and three of these he thought were doubtful. Cabot followed many cases of the classical triad of apical systolic murmur, loud pulmonary second sound, and an enlarged heart, but post-mortem there was rarely evidence of mitral incompetence without mitral stenosis. Mackenzie (1925) held almost similar views and regarded mitral stenosis as the essential rheumatic valvular lesion. On the other hand, de la Chapelle, Graef, and Rottino (1934) record the necropsy findings in 7 cases of mitral incompetence. Of 100 necropsies reported by Bland, White, and Jones (1935) on patients dying from rheumatic heart disease below the age of 21, 25 had valve deformity without stenosis: of these 100 cases, recrudescence of the rheumatic infection was directly responsible for 85 deaths. Dana and Reidy (1936) graded 150 cases of mitral disease at the post-mortem examination; pure mitral incompetence was not uncommon, forming in fact one-fifth to one-half of all cases; and the writers expressed the belief that this condition can by itself cause death. Levine (1945) believes that mitral incompetence is occasionally found post-mortem; he reports cases with apical systolic murmurs and subsequent bacterial endocarditis where post-mortem a pure incompetence of the mitral valve is found. Bourne (1946) has recently recorded the necropsy findings in such a case. It seems that pure mitral incompetence though uncommon does occur especially in the young, and proceeds to stenosis later, so that it is rarely seen without it after the age of twenty.

Twelve cases were seen in which doubtless there was mitral disease, but in which the evidence was insufficient to warrant the diagnosis of mitral stenosis. These were labelled mitral incompetence (rheumatic). All had an apical systolic murmur, 6 had a positive rheumatic history, 5 showed slight general enlargement on radioscopy, and 2 others slight enlargement of the left ventricle. Other and almost identical cases as regards signs (described above) were classed as mitral stenosis, mainly because of X-ray evidence.

A greater problem was presented by as many as 68 subjects (25 per cent of the whole group of mitral valvular disease) classed as " probable mitral valvular disease (mitral incompetence)." The alternative diagnosis was a healthy heart with a noticeable but incidental systolic murmur. An apology is perhaps necessary for the size of this group ; for in military practice it may be politic to exclude all who carry so suspicious a murmur lest later they should fall sick or claim unjustifiably a pension. In civilian life they might have been recorded as " For observation, ? organic heart disease " and would then have been followed up over a period of months or years. Here also phonocardiography will, in future, prove invaluable. Of this group, 45 gave a positive and circumstantial history of rheumatic fever or chorea in youth. Dyspnœa was claimed by 14, and 1 gave a history of hæmoptysis. A systolic murmur at the apex was heard in 45, varying from soft to moderate in intensity and short to moderate in length. In 24 of them the apical first sound was loud and sudden. In 9 doubt existed as to the presence of a mitral diastolic murmur. Of these cases 30 showed a doubtful or borderline enlargement of the left auricle in the right oblique position; in 8 the heart seemed to be generally though slightly enlarged; in 4 the cardiogram showed right axis deviation.

TABLE IV

CASES THOUGHT TO HAVE MITRAL VALVULAR DISEASE

Mitral stenosis	184
Mitral incompetence	12
Mitral valvular disease probable	68
	264

Mitral stenosis without murmur. In two cases the diagnosis rested on the radiological appearance alone. In one, auricular fibrillation was present and the heart was enlarged (X-ray), the size of the left auricle being diagnostic. The other case had tachycardia, and here the left auricle and the conus were both enlarged. Sosman (1940) has termed this variety the " sub-clinical type."

RADIOSCOPY IN MITRAL VALVULAR DISEASE

The difficulties of clinical diagnosis of mitral valvular disease have been reduced by the use of X-rays. In such inspections it is important to bear in mind the influence of the position of the diaphragm on the cardiac shadow. Just as a high diaphragm by raising the heart and rotating it may give an appearance similar to that of mitral stenosis, so a low diaphragm may conceal the minor changes to be expected in mitral disease.

Our radioscopic findings show no significant difference between the cardiac outline of mitral stenosis in those with a presystolic murmur and in those with a systolic murmur only. Among those with a presystolic murmur only, 104 in all, enlargement of the left auricle was the only certain X-ray change in 77 (74 per cent) ; in 13 the pulmonary artery and the conus were also enlarged; in a further 14 (13 per cent) there was no change in the size or shape of the heart. Of those with a diastolic murmur, 13 in all, 6 cases had a large left auricle, 4 enlarged pulmonary artery and conus; and 3 showed no change. Of those with an apical systolic murmur and mitral stenosis (60 in all), 40 (67 per cent) had left auricular enlargement, 14 had enlargement of the pulmonary artery and the conus, and a further 6 (10 per cent) showed no change. Thus about 10 per cent of cases of early mitral stenosis show no appreciable changes on radioscopy—a small percentage.

The first and commonest sign of mitral stenosis on the X-ray screen is enlargement of the left auricle; this occurred in about 70 per cent as the sole X-ray sign: it is seen best and earliest, in our opinion, in the conventional right oblique position with barium in the œsophagus. In the early case, enlargement is most noticeable with the subject turned through an angle of 60° rather than 45°; but the optimum angle should be chosen. In this position also the pulmonary artery, with perhaps the "conus" of the right ventricle, appears as a prominence on the anterior border of the heart, giving the appearance of great cardiac depth from front to back as compared with the slim-necked normal organ. Master (1942) considers the left oblique position to be of equal value ; and so does Evans (personal communication).

In the right oblique position the course of barium in the œsophagus in the normal is straight

or at most a gentle and slight arc (Fig. 1) ; but in early mitral stenosis, on reaching the upper border of the left auricle, the barium hesitates, *changes its course*, turning backwards before continuing in a curve around the auricle. The bend forming an obtuse angle varies in position, but it is usually high on the posterior cardiac border (Fig. 2, 3, and 4). A wedge of barium may be left at the site of the bend, filling the obtuse angle. Occasionally the convexity may affect not the upper but the middle part only of the auricular curve.

When the left auricular enlargement is but part of a general enlargement, or when this chamber is pushed backwards by a large left ventricle, the barium veers backwards at a high point and runs in a longer curve with its convexity dorsally; there is seldom such a sharp change in direction as in typical mitral stenosis.

An isolated or disproportionate enlargement of the left auricle is almost pathognomonic of mitral stenosis. Apart from a high diaphragm, there are other causes for backward displacement of the œsophagus, for instance, complete heart block (Babey, 1937), pericardial effusion, or patent ductus arteriosus (Donovan *et al.*, 1943). The question as to whether or not the left auricle may enlarge in long-standing auricular fibrillation without mitral stenosis is a matter of opinion; to us it seems probable, but to a limited degree (Babey, 1937; Palmer, 1937). Enlargement of this chamber also occurs in the heart of thyrotoxicosis, but it is more a part of a general enlargement of all chambers.

In the early case in which the right oblique shows a doubtful or borderline enlargement

FIG. 1.—Normal heart. Right oblique position with barium in the œsophagus. Normal left auricular curve.

FIG. 2.—Early mitral stenosis. Right oblique view with barium in the œsophagus. Increased cardiac depth. Barium changes course at a high level with wedging of the barium at the upper border of left auricle as the œsophagus is pushed backwards.

FIG. 3.—Early mitral stenosis. Description as for Fig. 2.

FIG. 4.—Early mitral stenosis. Description as for Fig. 2.

of the left auricle, a straightening of the left border in the anterior view as a separate consideration may assist in coming to the right conclusion on the presence or absence of an early lesion, for the anterior X-ray of mitral stenosis is often also characteristic. The earliest sign is a straightening due to filling-in of the normal concavity of this border by prominence of one or both of the two middle arcs (Fig. 5, 6, 7, and 8).

As some others have done, we deprecate the use of the term " mitralization " for this straightening of the normally concave left border of the heart. By such mixing of the premise and the conclusion in describing an X-ray picture, its real value is diminished. What is seen should be accurately described, and the interpretation for diagnostic purposes should logically follow. Thus straightening of the left border is the observation, and the probable but not the invariable interpretation is mitral stenosis. The upper of the middle arcs is admittedly formed by the pulmonary artery, with or without its left branch. Undue prominence of the upper middle arc (the pulmonary) is almost invariable in established mitral stenosis, but we have only seen it in a small proportion, say 10 per cent, of early cases.

The position as regards the lower middle arc is not so clear. Most cardiologists have believed and taught for years that this arc represents the enlarged conus of the right ventricle which extends forwards and to the left; and that, as the heart rotates to the left, it is thrown

FIG. 5.—Normal heart. Anterior view.

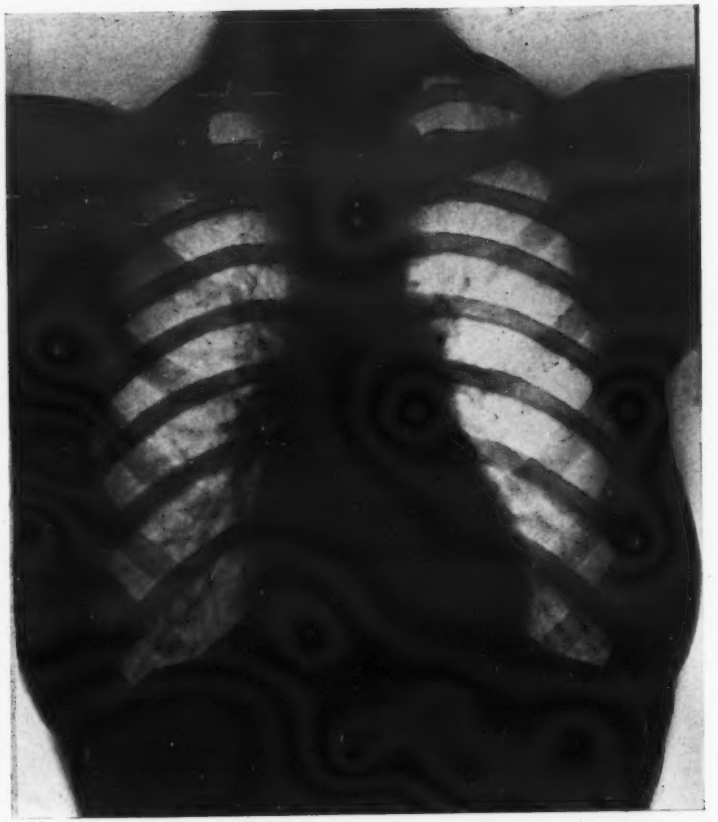


FIG. 6.—Early mitral stenosis. Anterior view. Straightening and lengthening of the left cardiac border by enlargement of the pulmonary artery and "conus."

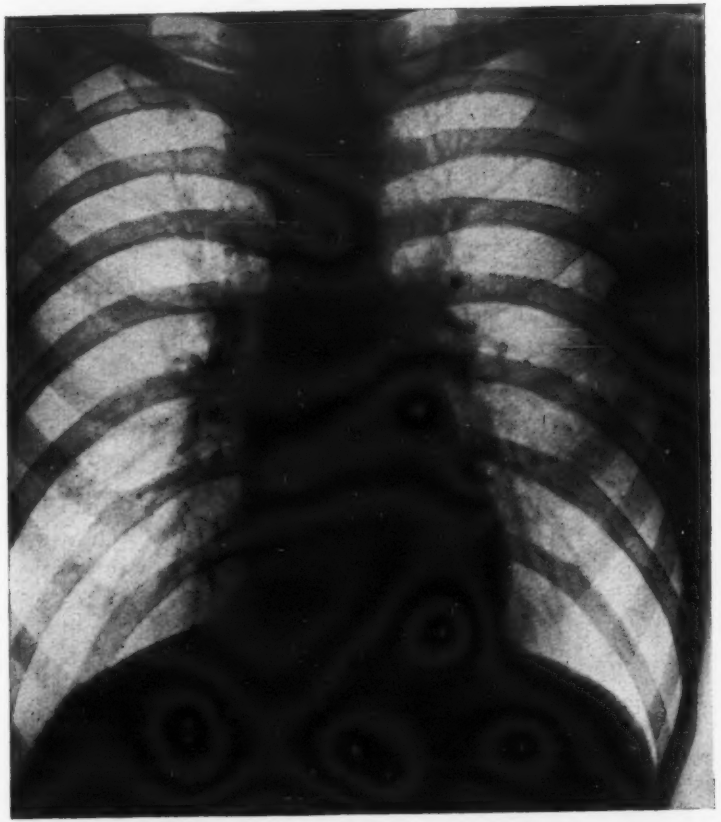


FIG. 7.—Early mitral stenosis. Anterior view. Straightening and lengthening of the left cardiac border by enlargement of the pulmonary artery and "conus."

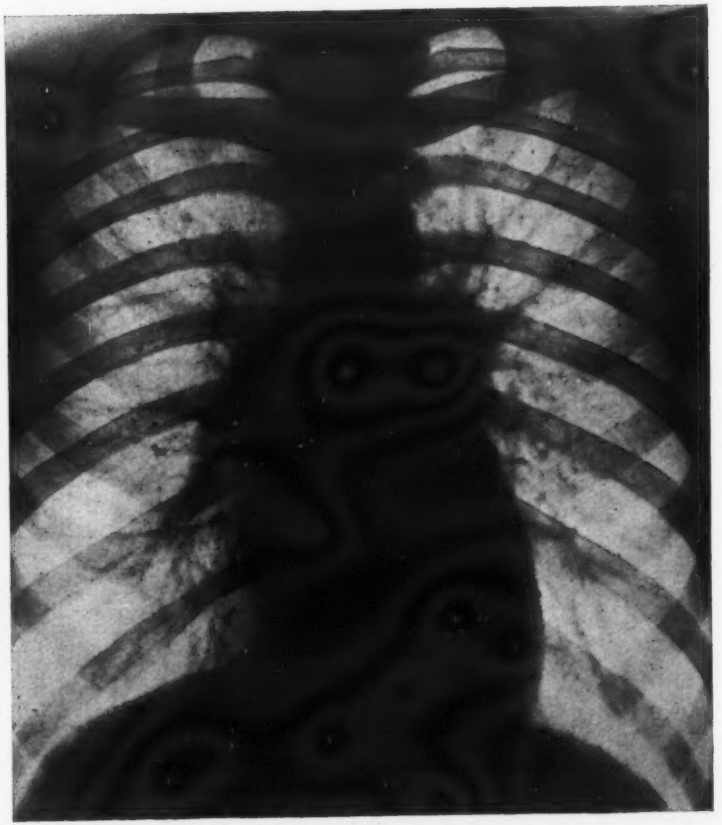


FIG. 8.—Early mitral stenosis. Description as for Fig. 7.

into more prominence. This teaching was founded on post-mortem study and found support from Laubry *et al.* (1939), and Roesler (1943). Robb and Steinberg (1939), and Grishman, Sussman, and Steinberg (1944), dispute this belief from their studies with angiocardiograms. They consider that this arc is formed by the left auricular appendage pushed into prominence by the left auricle enlarging to the left, not only to the right; they are convinced that the right ventricular conus never forms part of the cardiac outline as seen by X-ray. On the whole we accept this view.

Since reading this new conception of the identity of the lower middle arc we have taken every opportunity of noting whether while turning the patient to the left (right oblique position) the prominence disappears or persists, and we often find that it persists. If it were the left auricle or its appendage it might disappear with this rotation of the patient (see also Zdansky, 1939). This is not a valid objection to the newer view. The thickness of the ventricular wall is admittedly to be reckoned with. It may be that the undilated appendage sits upon the projecting conus augmenting the prominence. We do not know to what extent the auricular appendage is distended in life; at necropsy it is usually contracted.

Apart from invariable radioscopy which is part of our cardiac ritual, in order to study the anterior outline of the heart in the early stages of mitral valvular disease teleradiograms were taken of 32 men, 17 healthy students, 8 very early but certain mitral stenotics, and 7 men with mitral stenosis and aortic incompetence. This number is admittedly too small for statistical judgment, but they do give some indications of value. These 32 films were taken in 1943, but examined by each of us separately without reference to the clinical findings.

In 21 of the 32 films there was agreement as to the degree of *the hilar shadows*, in 10 of these 21 they were thought to be large or too dense, or both. Valvular lesions were present clinically in 7 of these 10, mitral stenosis with aortic incompetence in 4, mitral stenosis alone in 3. Among the 8 men with early mitral stenosis alone, there were increased hilar shadows in 3, in the other 5 we were not in agreement ; that is in no case of pure mitral stenosis were the hilar shadows considered to be normal by both observers. Among the 7 men with combined valve lesions it was agreed that 4 showed increased markings and that in 1 they were normal ; they was disagreement in 2 cases. The hilar shadows were thought to be normal by both observers in 9 of the 17 normal heart films, to be excessive in 3, and there was disagreement in 5 others. The conclusion may be drawn that the estimation of the degree of hilar markings carries a wide margin of error, yet increase in these shadows does occur rather more frequently in early valvular disease than in normal hearts.

The clinical diagnosis of early mitral stenosis had been made in 8 men ; one of us (J. P.) recognized mitral stenosis in all these films but thought that in 3 it was associated with aortic incompetence; the other (R. H.) considered that 3 of these hearts were within normal limits and also that aortic incompetence was added to the mitral stenosis in 2 others.

The outline of these 8 hearts with early mitral stenosis was traced from the teleradiograms and they were approximated; for comparison, similar tracings of 8 hearts from normal students are shown (Fig. 9). The difference seems to lie first in the straight left border of the group with mitral stenosis, and secondly in the small aortic knuckle in this group, these two points in combination giving an appearance of added length and straightness to the left border of the heart with mitral stenosis. The buttressing, i.e. extension outwards of the right auricle, was possibly a little more prominent in those with mitral stenosis than in those without it, or their right auricular curve may extend a trifle higher. Fig. 6, 7, and 8 show the earliest changes that we consider to be recognizable in mitral stenosis in the anterior view. Fig. 2, 3, and 4 show the earliest changes in the right oblique position. Examples of normal hearts are shown in Fig. 1 and 5 for comparison.

Of the films, 7 in number, of hearts with the combined lesion of mitral stenosis and aortic incompetence, one of us (J. P.) was correct in 5, diagnosed mitral stenosis alone in 1, and considered 1 to be within normal limits. The other (R. H.) thought that 6 showed the combined lesion and that 1 was within normal limits. Both thought one particular film to be that of a normal heart when it was not. The conclusion to be drawn from the films is that, while enlargement of the left auricle in the oblique position is the most certain early sign of mitral stenosis, the anterior film is often abnormal, and nearly always so if a combined valve lesion is present.

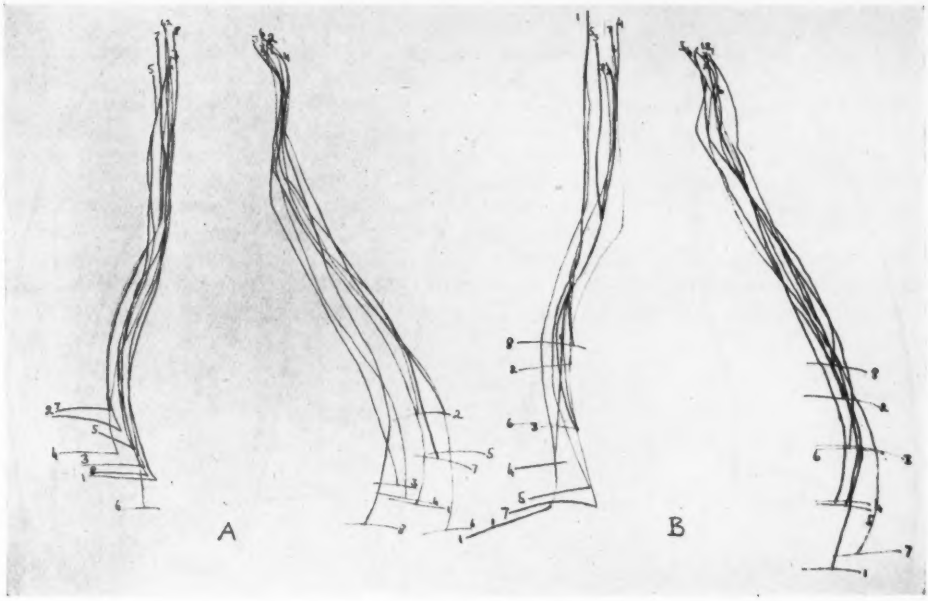


FIG. 9.—Diagram to compare the superimposed outlines of eight normal hearts (A) with those of eight early mitral stenotics; (B) in the anterior view. The difference lies in the straightening and lengthening of the left cardiac border. Buttressing of the right auricle is a less noticeable feature.

To illustrate those changes that we consider to be the earliest in mitral stenosis we have produced the diagrams shown in Fig. 10 and 11. A normal cardiac outline, taken from a teleradiogram, was used as the normal outline shown by the dotted lines. On to this we have drawn the abnormal outline that we believe to be typical of the early condition. This belief is founded on our experience of radioscopy in the early and symptomless case as seen in recruits.

The question of the differential diagnosis of mitral stenosis from mitral incompetence on radioscopy does not appear easy to decide. Dietlen (1923) considered mitral incompetence to be by far the commonest pure valve lesion seen ; he gave three points of difference from mitral stenosis in early cases : first, the left auricle is enlarged, but not to the same degree as in stenosis ; secondly, the right ventricle is not enlarged except in failure ; thirdly, the left ventricle is enlarged in incompetence, but not in stenosis. Assman (1934) says that there is hypertrophy and dilatation of all the heart chambers except the right auricle in mitral incompetence. Roesler (1943) considers mitral incompetence to be rare, and to cause enlargement of the left ventricle. He draws attention to the pulsations in the large left auricle; in mitral incompetence these are exaggerated and, as they alternate with those of the right lower border, cause a see-saw movement which may be quite distinctive. Zdansky (1939) also speaks of enlargement of the left ventricle, and of the left auricle, though not to the same degree as in mitral stenosis.

We have never felt that this differentiation between mitral stenosis and incompetence can be made radiologically with certainty until kymography is successfully applied to the problem. When, in the presence of a mitral systolic murmur, doubtful or certain enlargement of the left ventricle is seen, it is our practice to listen again for an aortic diastolic murmur. If in mitral incompetence the left ventricle enlarges, and if it is admitted that mitral incompetence develops at a later date into mitral stenosis, the question must arise why the left ventricle is not enlarged in mitral stenosis? Does the left ventricle regress as stenosis is installed ? This is hard to believe.

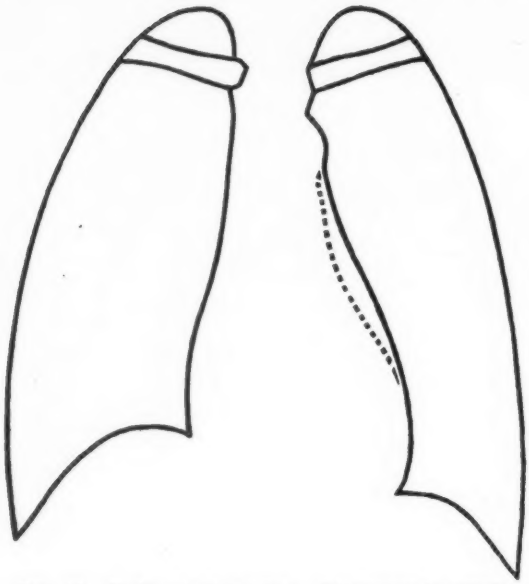


FIG. 10.—Early mitral stenosis. Diagrammatic anterior view. Dotted line denotes normal cardiac outline. Straightening of the left cardiac border by enlargement of the pulmonary artery and "conus," the earliest detectable change in this position.

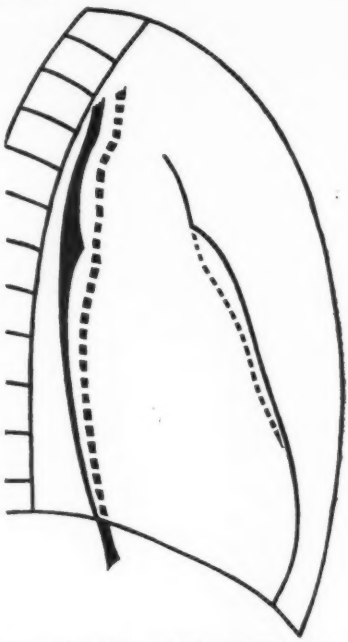


FIG. 11.—Early mitral stenosis. Diagrammatic right oblique view, with barium in the œsophagus. Dotted line indicates the normal heart outline and the normal œsophageal course. Increased cardiac depth, with left auricular enlargement. Anterior and upper cardiac border is fuller and more rounded than the normal.

AORTIC VALVULAR DISEASE

Aortic incompetence

There is remarkable unanimity as to what is the essential early sign of aortic incompetence; it is the aortic diastolic murmur. In early cases this murmur may be inconstant, present only within a limited range of heart rate, or in one position, or indeed on one occasion and not on another. It is usually heard best on the left of the sternum and follows immediately after the second heart sound.

In view of the high incidence rate of aortic diastolic murmurs heard best on the left of the sternum, it is of interest in surface anatomy to note that Sosman and Wosika (1934) have shown that, in disease at least, the aortic valve lies not behind the second right intercostal space, but behind the centre of the sternum, or sometimes to the left of the mid-line. Zdansky (1939) pictures the aortic valve in health to be just below the junction of the superior vena cava and the right auricle, and lying at the left border of the vertebral column in about the centre of the heart shadow at this level. Laubry *et al.* (1939) also indicate that this is the normal site of the aortic valves. Grishman *et al.* (1944) picture the aortic valve as being rather to the left of the mid-line.

Auscultation should be practised with the subject standing, sitting leaning forwards, and lying. As with mitral stenosis, listening to the patient lying down after exertion is useful, for an aortic diastolic murmur may best be heard during the lower ranges of a falling heart rate. The patient should always be asked to stop breathing for a moment at the end of expiration, for this excludes its noise and the respiratory sound which may closely resemble a diastolic murmur. Sometimes an aortic diastolic murmur is audible at the apex and then its early position in diastole, i.e. immediately after the second sound and before the normal time of the third heart sound, aids in its identification (Evans, 1942*a*).

That this murmur is frequently overlooked in recruits is confirmed by the report of Delaney *et al.* (1943) who examined 45,000 air cadets and detected heart disease in 100, 43 of whom showed aortic valvular lesions only; these were men who had been examined several times at medical boards and had been passed fit.

In our series, 202 cases of pure aortic incompetence of rheumatic origin were found. This large figure is significant in that it is a measure of the difficulty experienced by doctors in making this diagnosis. The diastolic murmur may be very difficult to detect unless it is listened for with specific intent, and with ears attuned. Too often an apical systolic murmur, present in 56 per cent of these cases, distracted undue attention upon the mitral valve to the neglect of the aortic and, therefore, to the detriment of the true diagnosis.

That aortic incompetence is frequently missed in life is also shown by a comparison between the clinical and pathological incidence (Tables V and VI). The variation among different authors probably rests on their different criteria of diagnosis in double valvular lesions.

TABLE V

THE VALVES AFFECTED (CLINICAL)

Name	No. of cases	Mitral	Aortic	Both
		Per cent	Per cent	Per cent
Hirschfelder, A. D. (1918)	1781	51	22	20
Willius, F. A. (1927)	160	78	13	9
Wilson, Lingg, and Croxford (1928) ..	328	88	0	11
Findlay, L. (1932)	489	88	0	12
Schlesinger, B. (1938)	738	87	1	11
Ritchie, W. T. (1939)	179	83	1	16
Sosman, M. C. (1939)	738	29	22	49

TABLE VI

THE VALVES AFFECTED (POST-MORTEM)

Name	No. of cases	Mitral	Aortic
		Per cent	Per cent
Coombs, C. F. (1924)	97	100	59
Clawson, B. J., *et al.* (1926)	130	73	63
Glahn, von W. C. (1927)	109	91	58
Findlay, L. (1932)	37	95	59
Bland, White, and Jones (1935)	100	98	71

An aortic diastolic murmur was present in all 202 cases. Dyspnœa was present in 31 (15 per cent compared with 18 per cent in mitral disease). It was recognized during the last war that pure aortic incompetence seldom gave rise to symptoms (Cotton, 1919). Bourne (1940) also found aortic incompetence to be asymptomatic more often than mitral stenosis.

A history of rheumatic fever was obtained in 84 (41 per cent), of chorea alone in 6 (3 per cent), and of both in 8 (4 per cent). The low incidence of aortic incompetence resulting from chorea has often been emphasized (Abt and Levinson, 1916; Strong, 1923; Wallace, 1933; Schwarz and Leader, 1935 ; and Berghoff *et al.*, 1944).

Peripheral vascular signs are absent in early cases ; capillary pulsation, collapsing pulse, and even a large pulse pressure on sphygmomanometry, are to be regarded as confirmatory signs in the developed disease, but render little service in earlier diagnosis.

The X-ray changes in aortic incompetence are confined to the left ventricle and to the aorta. The enlarged left ventricle moves the left border farther out, or the lower and apical portion becomes more rounded, or both these features are present (Fig. 12). In the second oblique position, with the patient turned through an angle of 45°–60°, the rounded left ventricle is in greater contrast with the straight right ventricular arc than in the normal (Fig. 13 and 14).

FIG. 12.—Early aortic incompetence. Anterior view. Left ventricle is enlarged to the left and also more rounded than is normal. Ascending aorta is slightly prominent for a young man.

The heart and aorta show excessive pulsation only if the reflux is great enough. A dynamic dilatation of the first part of the aorta is common in rheumatic aortic incompetence and should not easily suggest syphilis.

The diagrams in Fig. 15 and 16 show the changes that we consider to be the early radioscopic signs of aortic incompetence. Upon normal cardiac outlines in the anterior and left oblique positions, shown by the dotted lines, we have drawn that enlargement which our experience of the early and symptomless case, as seen in recruits, has lead us to consider to be the first typical change.

X-ray changes were present in 169 (84 per cent) of these 202 cases, and absent in 33 (16 per cent). The proportion with enlargement is noteworthy when it is remembered that these were early cases and the majority were asymptomatic. Aortic incompetence was strongly suspected in a further 40 cases which were returned as—rheumatic heart disease (probable aortic incompetence). In this group a combination of positive rheumatic history, symptoms, signs, and X-ray findings made the diagnosis likely, but no diastolic murmur was heard with any certainty. One such case was seen in 1942, and again one year later when a diastolic murmur was obvious.

Aortic incompetence of syphilitic origin was the lesion in a further 4 cases.

FIG. 13.—Normal heart. Left oblique view.

FIG. 14.—Early aortic incompetence. Left oblique view. Rounding and prominence of the left ventricle.

Aortic stenosis

Aortic stenosis alone was present in 14 cases. It is generally considered (Christian, 1931; Clawson *et al.*, 1938 ; Dry and Willius, 1939 ; and Reich, 1945) that aortic stenosis is mostly of rheumatic origin. Gibbs (1935) in his necropsy report found this to be so in 90 per cent under the age of 50, of 27 cases studied.

The diagnosis rests on the finding of an aortic systolic murmur and thrill, with left ventricular enlargement. Whether this diagnosis should be made in the absence of a thrill is debatable—we think it may be made. Reich (1945) recorded its presence in only 27 per cent of 22 cases confirmed post-mortem. A thrill was present in 13 of our 14 cases. The stenotic significance of a loud systolic aortic murmur as the main auscultatory sign in a person suspected of rheumatic heart disease cannot easily be discounted, and with left ventricular enlargement aortic stenosis would become the most likely diagnosis, thrill or no thrill. It is often stated that the aortic second sound is absent in aortic stenosis; but this is not true of early cases. In 13 of our 14 cases the second sound was heard; after the loud murmur, attention may then be directed to hearing the second sound. Analysing the 14 cases: 3 gave a rheumatic history, 6 an apical systolic murmur, and 12 exhibited left ventricular enlargement on radioscopy. Analysis of 13 cases of aortic stenosis with incompetence returned a rheumatic history in 1, dyspnœa in 2, an apical systolic murmur in 7, and X-ray enlargement of the left ventricle in all.

COMBINED VALVULAR LESIONS

Aortic incompetence and mitral stenosis

Aortic incompetence in combination with mitral stenosis was present in 72 cases (10 per

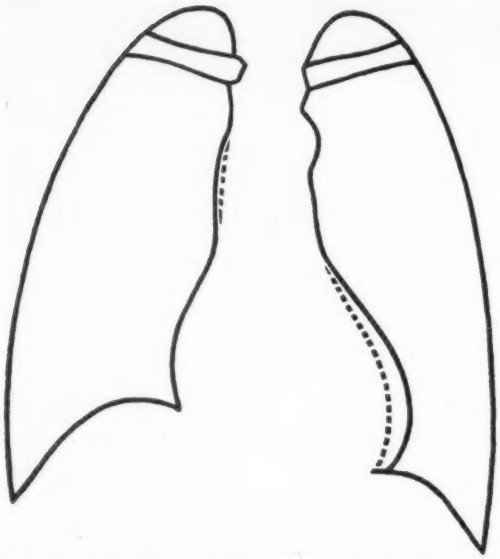


FIG. 15.—Early aortic incompetence. Diagrammatic anterior view. Dotted line is normal heart outline. Left ventricle enlarged to the left, and more rounded than the normal. Ascending aorta prominent.

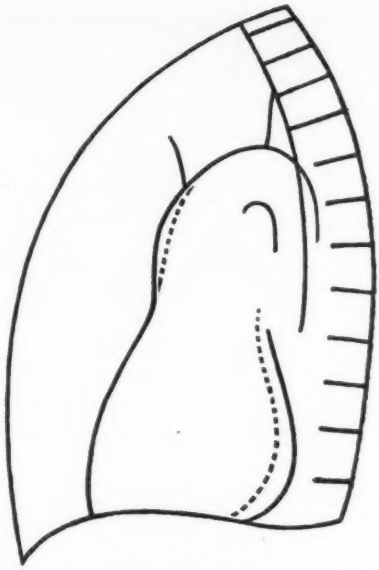


FIG. 16.—Early aortic incompetence. Diagrammatic left oblique view. Dotted line is normal heart outline. Left ventricle enlarged dorsad, and more rounded than the normal. Ascending aorta prominent.

cent) of rheumatic heart disease seen. All these seemed more fully developed than were the single valve lesions and no difficulty was found in their diagnosis.

It must be allowed that 10 per cent of rheumatic cases with this combined lesion does not represent the true figure in all rheumatic heart disease because the grosser cases were graded by the Boards without reference. We believe that this combination is far more frequent than is generally thought, and that the aortic murmur is simply overlooked. Table V shows the clinical frequency as shown by various reported series, and Table VI the post-mortem incidence, the difference between them being obvious.

The findings in our 72 cases are shown in Table VII.

TABLE VII

ANALYSIS OF 72 CASES OF MITRAL STENOSIS WITH AORTIC INCOMPETENCE

Rheumatic history		Dyspnœa	X-ray findings	
Rheumatic fever	23 (32 per cent)	21 (29 per cent)	Left auricle enlarged	53 (74 per cent)
Chorea	6 (8 „)		Left ventricle enlarged	45 (63 „)
Both	3 (4 „)		General enlargement	12 (17 „)
Total	44 „		Normal	4 (6 „)

Mitral stenosis, aortic incompetence, and stenosis were present in combination in four cases. A single example of mitral stenosis and aortic stenosis in combination was seen.

Hypertension

The problem of appraising the uppermost limits of the normal blood pressure remains unsolved; for the purpose of admission to the British armed forces a diastolic pressure of 90 must not be exceeded. The question of the significance of a transient hypertension has been much discussed, more in the United States than in this country (Hines, 1940 ; Master, 1943 ; Rogers and Palmer, 1944; and Levy *et al.*, 1944). The conclusions drawn by these

workers is that men showing it, and those with a borderline reading, are more likely to proceed to true hypertension than those without it.

In our 2500 recruits, only 49 (2 per cent) were seen with hypertension, i.e. 6 per cent of all those with heart disease. As noted above, this figure is not a true representation of the frequency of hypertension as compared with rheumatic heart disease. In 45 the systolic and diastolic pressures were both raised, in 2 the systolic only, and in a further 2 the diastolic only. The figure of 160/90 was taken as borderline. The average reading for all these cases was 176/104.

We think that the upper limit of the systolic pressure (after the exclusion of renal disease and coarctation of the aorta) might with safety be placed higher than 160 mm., provided that the diastolic pressure is 90 or below, and that there is no evidence of left ventricular enlargement on radioscopy.

Changes of Pulse Rate

Tachycardia. A simple nervous tachycardia seemed worthy of note in 72 recruits. The pulse ranged between 90 and 130, but usually it fell after rest and as the man became more accustomed to his examiner.

In the United States a persistent heart rate of 100 or over is a cause for rejection (White, 1943). Hoskin (1942) considers that nervous tachycardia should be graded low for service. We believe that a slightly more liberal attitude should be adopted, a rate of 120–130 which tends to fall on resting, in the absence of thyroid disease, may be accepted for full service. We would agree that a persistent rate of 100 should lead to a careful search for a cause other than simple nervousness, such as pyrexia, pulmonary disease, and thyrotoxicosis. Excessive smoking or consumption of alcohol may accelerate the pulse rate.

Bradycardia. This is seldom worthy of note. When the rate is under 50 an attempt to raise the pulse by exercise should be made; when under 40 an electrocardiogram is indicated. Parkinson (1936) has emphasized that simple bradycardia may produce a natural enlargement of the cardiac shadow on X-ray. Two such cases were seen; one because a radiological opinion had been sent to the medical board previously that the heart was enlarged; the other because of the co-existence of a soft systolic murmur.

In 1000 consecutive cases, 31 were referred for irregularity of the pulse.

Extrasystoles	16
Sinus arrhythmia	10
Auricular fibrillation	3
Auricular flutter	1
Tachycardia with ventricular escape	1

In all, 5 cases were seen with auricular fibrillation of undetermined ætiology.

Cardiac enlargement

A difficult problem arose in 25 recruits in whom the heart was enlarged on X-ray, but the cause could not be found. In view of the age group they must be regarded as either congenital or rheumatic. We believe that some of these will eventually develop an aortic diastolic murmur, and this we saw to occur in one case. A further 41 were referred on account of an apparent enlargement from a displaced apex-beat; but actually there was no enlargement, and the displacement was due to scoliosis, which was often more verified on radioscopy than on clinical inspection.

Congenital heart disease

Among the 2500 men seen, 71 had congenital heart disease compared with 609 with rheumatic heart disease.

Pulmonary stenosis	21
Patent ductus arteriosus	10
Atrial septal defect	9
Ventricular septal defect	8
Aortic stenosis	2
Congenital dextrocardia	1
Coarctation of the aorta	1
Right-sided aortic arch	1
Unclassified	18
	71

Finally we would like to support a recent statement by Moir and Shirley Smith (1946) that " the remarkable low incidence of cardiovascular disorders in the Army (less than 7 per 1000 of all sick admitted to hospital) reflects much credit on Recruiting Medical Boards who succeeded in eliminating at the early stage the vast majority of patients with organic cardiovascular disease and to a large extent weeding out those prone to effort syndrome and other neuro-circulatory upsets."

Summary and Conclusions

Medical Boards referred 2500 recruits to one of us for a cardiological opinion before acceptance for military service, and of these, 609 (24 per cent), about a quarter, were judged to have chronic rheumatic valvular disease. The commonest of reasons for reference was the presence of a systolic murmur; others were displacement of the apex-beat, an altered first heart sound, a noticeable third heart sound, arrhythmia, and tachycardia.

Incidental (or functional) murmurs were heard in 259 men (10 per cent). The basis for recognition was their softness and shortness, their variability with posture, respiration, and heart rate. An incidental murmur is seldom loud or long, unless it is of the " late-systolic " variety. A heart found to be of normal shape and size on radioscopy was valuable, and corroborative evidence in deciding that a murmur was incidental. Military as well as medical reasons will decide whether the occasional recruit with a noticeable yet innocent murmur should be accepted for war service.

A split *first-sound* at the apex is a normal variation often mistaken for a presystolic murmur; and slight modifications in the quality of the first sound are negligible. Only in the absence of tachycardia is a loud and abrupt first sound suggestive of mitral stenosis.

Mitral valvular disease was present in 264 men, 32 per cent of all rheumatic cases (609). Pathological evidence is quoted to support the view that mitral incompetence, without stenosis, must occur clinically though it is rare except perhaps in early life. If there is doubt the term mitral valvular disease is convenient.

(*a*) *Mitral stenosis with a presystolic murmur.* 104 cases; all had a loud first sound. In 77 cases enlargement of the left auricle was the characteristic X-ray change: 5 other cases had a loud first sound without a presystolic murmur, and yet X-rays left no doubt that mitral stenosis was present. A mitral diastolic murmur only was heard in 13 other cases.

(*b*) *Mitral stenosis with a systolic murmur.* 60 cases; strong rheumatic history in over half, moderately or very long and loud murmur in all, loud first sound in some and X-ray evidence in 54. In 9 of the 60 cases there was a systolic thrill. In 12 other cases, similar evidence supported in 7 by X-ray enlargement of the heart (though not of the left auricle) seemed to justify a diagnosis of rheumatic mitral incompetence.

(*c*) *Mitral valvular disease* (probable). 68 cases ; the history, symptoms, and signs together strongly favoured the diagnosis, but they were not conclusive. In half of them, our X-ray note was " doubtful enlargement of the left auricle." Rejection was always advised.

(*d*) *Mitral stenosis without murmur.* In two cases, one with auricular fibrillation, no abnormal auscultatory signs were found, but the enlargement of the left auricle justified the diagnosis of mitral stenosis.

Aortic valvular disease, without mitral stenosis was present in 229 men.

(*a*) *Aortic incompetence alone.* 202 cases ; all had an aortic diastolic murmur; more than half had also an apical systolic murmur of no great length or intensity. In none was the left auricle enlarged, but in 169 cases (84 per cent) there was enlargement of the left ventricle.

Aortic incompetence alone or in combination with mitral stenosis is far commoner than usually thought. Pathological evidence is quoted in support of our belief. During routine auscultation the possibility of aortic incompetence should always be born in mind, and especially perhaps where there is a systolic murmur at the apex otherwise unexplained.

(*b*) *Aortic stenosis alone.* 14 cases ; 13 had an aortic systolic thrill and yet the aortic second sound was present ; 12 showed enlargement of the left ventricle.

(*c*) *Aortic stenosis and incompetence.* 13 cases ; all had enlargement of the left ventricle.

Combined valvular lesions. Aortic incompetence and mitral stenosis. 72 cases, i.e.

10 per cent of all rheumatic valvular disease seen. Aortic stenosis also was present in 4 other cases.

Radioscopy

This proved to be invaluable in early diagnosis, but electrocardiography was of little value. In no less than 80–90 per cent of these early cases convincing X-ray evidence in support was found.

In the right oblique position the abnormal auricular curve is perhaps seen better on radioscopy than in a film as the course of barium in the œsophagus may be followed; this method also allows the subject to be turned until the optimum position is reached. In the right oblique view the anterior cardiac border in mitral stenosis is prominent and the normal slim neck of the heart becomes filled in and convex.

Left auricular enlargement in mitral stenosis and left ventricular enlargement in aortic incompetence are easily the most important early changes. The enlarged left auricle is confirmed as a sign equal in importance to the presystolic murmur in the diagnosis of mitral stenosis.

Diagrams are shown to illustrate those X-ray changes that our experience in radioscopy of the early and symptomless case has led us to accept as the early signs of mitral stenosis and of aortic incompetence.

A high diaphragm, a trivial scoliosis, or a simple bradycardia simulate (in radiology) an early mitral stenosis or an early aortic incompetence. Either lesion may be concealed by a low diaphragm.

In early mitral stenosis the filling in of the left border combined with a small aortic knuckle, gives a characteristic appearance of added length and straightness to this border. Extension upwards of the right border (buttressing) is unconvincing as an early sign. The density and extent of hilar markings vary so much in health that the occasional increase seen in early mitral stenosis has little value in diagnosis.

In early aortic incompetence the left ventricle enlarges as is shown both by extension outwards of the left lower border and by increased convexity of it. These together are regarded as the earliest signs of left ventricular enlargement. This enlargement is best confirmed in the left oblique position.

Confirmatory teleradiograms were taken in a small series of early valvular disease and in controls, and a few are reproduced in illustration.

We wish to thank Dr. William Evans of the Cardiac Department of the London Hospital for his help in revising this paper; Dr. Alastair Hunter, formerly Chief Assistant in the Cardiac Department, for the normal teleradiograms used as controls; and Dr. John Grimshaw for his valued help with the text.

References

Abt, I. A., and Levinson, A. (1916). *J. Amer. med. Ass.*, **67,** 1342.
Assman, H. (1934). "Die klinische Röntgendiagnostik der inneren Erkrankungen," Berlin, 5th ed., pp. 66, 77.
Babey, A. (1937). *Amer. Heart J.*, **13,** 228.
Baker, L. A., Sprague, H. B., and White, P. D. (1943). *Amer. J. med. Sci.*, **206,** 31.
Berghoff, R. S., Geraci, A. S., and Hirsch, D. A. (1944). *Med. Clin. North Amer.*, **28,** 86.
Bland, E. F., and Jones, T. D. (1938). *Arch. intern. Med.*, **61,** 161.
——, White, P. D., and Jones, T. D. (1935). *Amer. Heart. J.*, **10,** 995.
Blumenthal, J. S. (1942). *Ann. intern. Med.*, **17,** 637.
Bourne, G. (1940). *Brit. med. J.*, ii, 442.
—— (1946). *Ibid.*, i, 143.
Bramwell, C. (1943). *Brit. Heart J.*, **5,** 24.
—— (1942). *Glasgow med. J.*, **137,** 1.
Cabot, R. C. (1926). "Facts on the Heart." Phila. and London, p. 289.
Chapelle, C. E. de la, Graef, I., and Rottino, A. (1934). *Amer. Heart J.*, **10,** 62.
Christian, H. A. (1931). *J. Amer. med. Ass.*, **97,** 158.
Clawson, B. J., Bell, E. T., and Hartzell, T. B. (1926). *Amer. J. Path.*, **2,** 193.
——, Noble, J. F., and Lufkin, N. H. (1938). *Amer. Heart J.*, **15,** 58.
Contratto, A. W. (1943). *New Engl. J. Med.*, **228,** 499.
Coombs, Carey F. (1924). "Rheumatic Heart Disease," Bristol, p. 58.
Cotton, T. F. (1919). *Lancet*, **2,** 470.
Dana, H. W., and Reidy, J. A. (1936). *Amer. J. med. Sci.*, **191,** 109.
Delaney, J. H., Miller, S. I., Kimbro, R. W., and Bishop, L. F. (1944). *J. Amer. med. Ass.*, **123,** 884.
Dietlen, H. (1923). "Herz und Gefässe im Röntgenbilde." Leipzig, p. 189.
Donovan, M. S., Neuhanser, E. B. D., and Sosman, M. C. (1943). *Amer. J. Roentgen.*, **50,** 293.
Dry, T. J., and Willius, F. A. (1939). *Amer. Heart J.*, **17,** 138.

Evans, W. (1942*a*). *Brit. Encyl. Med.*, Pract. Surveys and Abstracts Volume, pp. 44–47, London.
—— (1942*b*). *Brit. Heart J.*, **5**, 205.
Fenn, G. K., Kerr, W. J., Levy, R. L., Stroud, W. D., and White, P. D. (1944). *Amer. Heart J.*, **27**, 435.
Findlay, L. (1932). "Rheumatic Infection in Childhood." London, pp. 74, 77.
Freeman, A. R., and Levine, S. A. (1933). *Ann. intern. Med.*, **6**, 1371.
Gibbs, A. J. (1935). *Guy's Hosp. Rep.*, **85**, 275.
Glahn, W. C. Von (1927). *Arch. Path. and Lab. Med.*, **3**, 355.
Grishman, A., Sussman, M. L., and Steinberg, M. F. (1944). *Amer. J. Roentgen.*, **51**, 33.
Hill, I. G. W., and Dewar, H. A. (1945). *Lancet*, **2**, 161.
Hines, E. A. (1940). *J. Amer. med. Ass.*, **115**, 271.
Hirschfelder, A. D. (1918). "Diseases of the Heart and Aorta." Phila. and London., 3rd ed., p. 398.
Hoskin, T. J. (1942). *Postgrad. med. J.*, **18**, 3.
Laubry, C., Cottenot, P., Routier, D., and Heim de Balsac, R. (1939). "Radiologie clinique du Coeur et des gros vaisseaux." Paris, Vol. I, pp. 23, 88, 89.
Levine, S. A. (1933). *J. Amer. med. Ass.*, **101**, 436.
—— (1945). "Clinical Heart Disease." Phila. and London, 3rd ed., pp. 31, 32.
—— and Likoff, W. B. (1944). *Ann. intern. Med.*, **21**, 298.
Levy, R. L., Hillman, C. C., Stroud, W. D., and White, P. D. (1944). *J. Amer. med. Ass.*, **126**, 829.
——, Stroud, W. D., and White, P. D. (1943). *Ibid.*, **123**, 937, 1029.
Mackenzie, Sir James (1925). "Diseases of the Heart." London, 4th ed.
Master, A. M. (1942). "The Electrocardiogram and X-Ray Configuration of the Heart." London, 2nd ed., p. 153.
—— (1943). *Bull. N.Y. Acad. Med.*, **19**, 704.
—— (1944). *U.S. Nav. Med. Bull.*, **42**, 307–15.
Moir, R. A., and Smith, K. Shirley (1946). *Brit. Heart J.*, **8**, 110.
Palmer, J. H. (1937). *Med. Res. Council*, Spec. Rep. Series, No. 222, London.
Parkinson, J. (1936). *Lancet*, **1**, 1337, 1391.
—— (1945). *Ibid.*, **2**, 657.
Perrot, G. St. J. (1944). *Milbank mem. fund. quart.*, **22**, 358.
Reich, N. E. (1945). *Ann. intern. Med.*, **22**, 234.
Ritchie, W. T. (1939). *Lancet*, **2**, 581.
Robb, G. P., and Steinberg, I. (1939). *Amer. J. Roentgen*, **42**, 14.
Roesler, H. (1943). "Clinical Roentgenology of the Cardiovascular System." 2nd ed., pp. 274, 283, Springfield, Ill.
Rogers, W. F., and Palmer, R. S. (1944). *New Eng. J. Med.*, **230**, 39.
Rowntree, L. G. (1945). *J. Pediat.*, **26**, 220.
Scadding, J. G., and Wood, P. (1939). *Lancet*, **2**, 1208.
Schlesinger, B. (1938). *Ibid.*, **1**, 593.
Schwarz, H., and Leader, S. D. (1935). *Amer. J. Dis. Child.*, **49**, 952.
Sosman, M. C. (1939). *Amer. J. Roentgen.*, **42**, 47.
—— (1940). *J. Amer. med. Ass.*, **115**, 1061.
—— and Wosika, P. H. (1934). *Amer. Heart J.*, **10**, 156.
Steell, Graham (1906). "Text Book on Diseases of the Heart." Manchester, p. 121.
Strong, G. F. (1923). *Canad. med. Ass. J.*, **13**, 92.
Thayer, W. S. (1909). *Arch. intern. Med.*, **4**, 297.
Wallace, H. L. (1933). *Edin. med. J.*, **40**, 417.
White, P. D. (1927). *Amer. J. med. Sci.*, **174**, 731.
—— (1943). *Ann. intern. Med.*, **18**, 323.
Wilburne, M., and Ceccolini, E. M. (1944). *Ann. J. med. Sci.*, **207**, 204.
Willius, F. A. (1927). *Amer. Heart J.*, **3**, 139.
Wilson, M. G., Lingg, C., and Croxford, G. (1928). *Ibid.*, **4**, 164.
Wood, F. C., Wolferth, C. C., and Miller, T. G. (1941). *War Medicine*, **1**, 696.
Zdansky, E. (1939). "Röntgendiagnostik des Herzens und der grossen Gefässe." Vienna, pp. 159, 164.

PROCEEDINGS OF THE CARDIAC SOCIETY OF GREAT BRITAIN AND IRELAND

The TENTH ANNUAL GENERAL MEETING of the Cardiac Society of Great Britain and Ireland was held at the Physical Chemistry Laboratory, Oxford, on Thursday, April 11, 1946. Chairman F. G. HOBSON.

The Chairman took the Chair at 10.0 a.m.; 64 members and 10 visitors were present.

PRIVATE BUSINESS

1. The minutes of the last meeting having been printed in the Journal (7,210, 1945) were approved and signed.

2. The accounts, audited by East and Brown, were approved: they showed a balance of £17 8*s.* 5*d.* The Council had decided that the ordinary subscription should be collected for the year 1946/47.

3. Maurice Campbell having completed the five years for which he was elected Secretary and five additional years during the war, Shirley Smith was, on the nomination of the Council, elected Secretary, to take office after the conclusion of the present meeting.

4. The following new members were elected:—

Ordinary Members	*Associate Members*
R. Ellis	R. Kempthorne
Evan Jones	Peter Kerley
A. Morgan Jones	D. Lewes
J. D. Olav Kerr	H. E. S. Pearson
W. Phillips	W. Stokes

Eight Associate Members were re-elected for another period of three years.

5. Geoffrey Bourne, London, and J. H. T. Towers, Leeds, were elected members of the Council for the years 1946–50.

6. The following changes in the Rules were carried, *nem. con.*, on the recommendation of the Council:

Rule 1. The Society shall be called the British Cardiac Society.
Rule 4. Insert Corresponding Members.
Rule 6. Ordinary Members shall not exceed 75 (instead of 60).
Rule 9. Associate Members shall not exceed 50 (instead of 30).
. . . not more than 20 instead of 15 shall be allowed in any year.
Rule 15 (New). Corresponding Members may be elected on the nomination of the Council from recognized cardiologists in the British Commonwealth or abroad.

7. The Secretary reported that on the instructions of the Council he had written to the president of the Royal College of Physicians with reference to (i) the appointment of representatives on the Consultant Services Committee, and (ii) the absence of any suggestions for Cardiology in the present list of specialties in a Consultant Service for the Nation. The Society decided to appoint William Evans as their representative on the Consultant Services Committee, and instructed the Secretary to write to the President of the College with regard to the formation of a standing Committee of the College to deal with Cardiology on the same lines as the Committees appointed for other specialties such as Neurology and Dermatology.
8. Correspondence with the Secretary of the Association of Physicians was reported. The possibility of some joint meetings was favoured and the Secretary was instructed to reply accordingly.
9. Williams Evans was again asked to act as Recorder.
10. Cardiac Emergencies was chosen for the main discussion at next year's meeting and the Secretary and Secretary-elect were deputed to divide up the subject and choose opening speakers.

DISCUSSION ON THE TREATMENT OF BACTERIAL ENDOCARDITIS WITH PENICILLIN

R. V. CHRISTIE (*introduced*) described the results of the team of workers who had been treating infective endocarditis with penicillin. Preliminary results had been published in the

Brit. Med. J. (1946, **1**, 881) but he was able to add to the number of cases and to follow them for rather longer.

He concluded that the duration of treatment was most important and the course should be continued for 28 days; that half a million units a day appeared an adequate amount to give; and that heparin should not be added. The occurrence of fever was not a reason for continuing treatment longer. Relapses, if they occurred, were generally in the first 10 days and nearly always in the first 30 days. Relapse after a short course did not seem to prevent later success, but relapse after a long course mattered more. After a relapse the same amount should be given for 6–8 weeks.

The sensitivity of the organism to penicillin did not matter, except that if it was 10 or 16 times more resistent than the standard, the cures were 30 per cent instead of 60 per cent.

The prognosis was better in congenital than in rheumatic heart disease. Severe heart failure was of grave import and 90 per cent died. The state of nutrition was also important and treatment within the first 6 months of onset was favourable.

Heart failure was the main cause of death, and emboli were also important.

G. E. S. WARD described 19 cases of bacterial endocarditis in which a blood culture showed *Streptococcus viridans*, and where 60,000 units of penicillin were injected intramuscularly at three-hourly intervals. Two of the 9 cases given penicillin for 21 days relapsed and had the same dosage for another 28 days. The remaining cases were treated for 28 days and none relapsed. Four of the 19 died, but only one of these could be regarded as having failed to respond to penicillin therapy, for in one cerebral embolism took place on the second day; another died of hæmorrhage from œsophageal veins in hepatic fibrosis; a third died from rupture of an aortic cusp. Of the 15 surviving patients, at least 11 are apparently cured and the other 4 may be. Two have been well for over a year, 5 for almost a year, 4 for nine months and 4 for shorter periods. One had congestive heart failure on two occasions but recovered. Dental extraction appeared to have precipitated the infection in 3 cases, and 9 others had dental sepsis (*Streptococcus viridans*); these 9 had teeth extracted under cover of additional injections of penicillin for 48 hours. Ward emphasized the danger of dental sepsis in rheumatic and congenital heart disease, and the importance of penicillin therapy before dental extraction in such cases. After reviewing the clinical features of the series he said that penicillin sensitivity gave no real clue to the prospect of success or failure, but that the number of colonies grown on culture media seemed to have a bearing on prognosis. Early and prolonged treatment, with adequate dosage of not less than half a million units of penicillin daily for 28 days, was essential; and in the case of a relapse, a further course lasting six weeks should be given.

HENRY COHEN (*introduced*) reported that the Liverpool findings closely paralleled those of other centres. Of 20 unselected consecutive cases (12 m. 8 f.) treated with penicillin, 8 (3 m. 5 f.) patients had died; the remaining 12 had survived for periods ranging from a few weeks to over a year. All three congenital heart cases had responded well. Patients preferred three-hourly intramuscular injection to a continuous intramuscular drip. Cohen spoke of the risks of major emboli, and made reference to a case of aneurysm of the left renal artery which was recognized and successfully treated by nephrectomy, several months after the endocardial infection had subsided. He stressed the importance of appreciating that dangerous sequelæ needing surgical intervention may follow the recovery from infection and the consequent prolongation of life. Another patient had been re-admitted to hospital with rapidly fatal heart failure due to rupture of an aortic cusp recurring long after the infection was overcome, and as the autopsy revealed the endocarditis was healed. Suggestive evidence was forthcoming, though a large scale investigation was necessary for proof, of the value of 100,000 units of penicillin daily for 2 to 3 days following dental extraction or tonsillectomy in a cardiac patient. There must always remain a proportion of cases—probably about 25 per cent who would succumb in spite of adequate penicillin therapy, from congestive heart failure, nephritis, major emboli, ruptured valves and aneurysms, and endocardial reinfection with a penicillin-resistant organism or strain.

GILCHRIST, BOURNE, BRUCE PERRY, and MORGAN JONES also recounted their experiences with a series of cases treated with penicillin.

SHORT COMMUNICATIONS

THE ACTION OF THEOPHYLLINE-ETHYLENE-DIAMINE IN HEART FAILURE

S. HOWARTH (*introduced*), J. MCMICHAEL, and E. P. SHARPEY-SCHAFER found that the theophylline component of theophylline-ethylene-diamine lowered venous pressure and increased cardiac output in normal hearts. The venous-pressure-lowering action was more persistent than the stimulating action on the heart, the latter being transient and often passing off within half an hour. Striking rises in cardiac output occurred in hypertensive heart failure, but the effects were less prominent in mitral stenosis.

THE HEART IN DEPRESSION OF THE STERNUM

BY WILLIAM EVANS

(Published in full, 1946, 8, 162.)

CHRONIC DISSECTING ANEURYSMS

BY A. MORGAN JONES and F. A. LANGLEY

(Published in full, 1946, 8, 191.)

BERNHEIM'S SYNDROME

TERENCE EAST described three cases in whom the heart failure was of the right ventricular type, although the lesion affected the left ventricle.

The first case was a man of 30 with aortic stenosis. There was the usual congestive failure, but the circulation rate was not greatly slowed. Post-mortem, the aortic valves were calcified, the left ventricle bulged into the right and reduced its capacity, and the lungs were free from engorgement and œdema.

The second case was that of a woman of 47 with aortic stenosis. Post-mortem, the valves were calcified, the lungs were free from œdema and engorgement, the septum bulged into the right ventricle, and there was general anasarca.

The third case was a man with hypertension who had begun to develop signs of right ventricular failure, the lungs remaining clear and the circulation rate not being much slowed.

Casts of the right ventricle in the first two cases were shown illustrating its diminished capacity.

Bernheim's syndrome may be expected in a case with a lesion affecting the left ventricle in whom failure of the right side develops, the circulation time not being unduly prolonged and the lungs remaining relatively clear.

DEMONSTRATION OF INTERATRIAL SHUNTS BY CARDIAC CATHETERIZATION

E. P. SHARPEY-SCHAFER and J. MCMICHAEL said that catheterization allowed the estimation of the oxygen unsaturation of blood in the right ventricle, right auricle, and superior and inferior venæ cavæ. In left to right shunts auricular blood was less unsaturated than caval blood. Estimation of arterial samples indicated right to left shunts. A case was also shown in which other evidence suggested an interatrial shunt but catheterization showed that a shunt was not present.

WOUNDS OF THE HEART

PAUL WOOD drew attention to delayed and recurrent attacks of pericarditis with or without effusion as a sequel to pericardial foreign body.

UNIPOLAR PRÆCORDIAL AND LIMB LEADS

C. W. CURTIS BAIN discussed unipolar chest leads. Standard leads are bipolar, and represent about equally the potentials of the two extremities connected to the galvanometer. With præcordial leads the influence of the remote electrode is very much less than that of the præcordial electrode, since it is so much farther from the heart; but any effect all from this electrode must be regarded as distortion. This applies especially when the potentials

at one point on the præcordium are to be compared with those at another. Wilson in 1932 devised a means of obtaining a remote electrode at almost zero potential, by connecting all three limbs to the galvanometer through a central terminal, and using this as the remote electrode. By this method, according to the Einthoven triangle hypothesis, all forces parallel to the plane of the triangle are cancelled out. There are also forces perpendicular to this plane and these impart a slight negativity to the tracings, but they are probably nearly constant throughout the cardiac cycle, and do not exceed 0·3 mv. For practical purposes these leads (V leads) can be considered to be unipolar. Wilson interposed resistances between each limb and the central terminal, but Goldberger in 1942 showed that they were unnecessary.

When using unipolar leads it is possible to obtain the potentials at any point on the body. Unipolar limb leads, reflecting the potentials at the right arm, left arm, and left leg, are useful in showing the position of the heart. The heart becomes more vertical when there is right-sided hypertrophy or a low diaphragm from emphysema or a long narrow chest. The heart becomes more horizontal when the left ventricle is hypertrophied or when the diaphragm is is high. Six præcordial leads are taken, as standardized by the American Heart Association (1938), extending from the right of the sternum to the mid-axilla. In a normal series the height of R in leads over the right ventricle (V1 and V2) is about half the depth of S; S is absent in leads over the left ventricle (V5 and V6). The transitional point, where R and S are equal, occurs about V3, which lies over the position of the normal septum.

The diagnosis of ventricular hypertrophy is based on the relative time taken by the impulse to spread through each ventricle. It is, therefore, unaffected by the position of the heart. In left ventricular hypertrophy R becomes small and S deep in leads V1 and V2; the voltage of the complexes usually increases; the transitional point swings to the left; in advanced cases T is inverted in V5 and V6. In right ventricular hypertrophy a Q wave appears followed by a large R in V1 and V2; there is no S. In V5 and V6, S waves are seen, but there is no abrupt transitional point.

When left-sided weakness is followed by right-sided hypertrophy, as in many cases of advanced hypertension, standard leads usually fail to show axis deviation, since the vertical twist of the heart caused by the right-sided hypertrophy cancels out the left axis deviation. Chest leads, however, continue to register the lengthened time taken by the impulse to pass through the thickened left ventricle. At the same time unipolar limb leads will show that the heart is vertical.

Chest leads in bundle branch block have the same general characteristics as in hypertrophy, but the QRS is widened. In left branch block a deep S follows a diminutive R in V1 and V2; in V5 and V6 a broad R occurs, and there is no S. In right branch block a broad R, sometimes preceded by a Q, is seen in V1 and V2, and a slender early R followed by a broad S in V5 and V6. Concordant and homophasic curves are shown to be due only to unusual positions of the heart. It is nearly always possible to determine the side of the lesion.

Anterior infarcts can be divided into antero-septal and antero-lateral types, according to whether the changes are more pronounced towards the right or left. Antero-septal infarcts often do not cause characteristic changes in lead I : small ones may only show inversion of T in V1, V2, and V3, and so would be missed if lead IV only were taken. Large anterior infarcts have deep Q waves and inversion of T in all the chest leads. Involvement of the septum can sometimes be diagnosed. Left branch block obscures the changes of infarction, since the left ventricle receives the impulse late. But this does not apply to right branch block, and the characteristic changes of branch block and the anterior infarct can both be seen clearly.

Heart Failure of Unknown Aetiology in Africans

D. Evan Bedford and G. L. S. Konstam (*introduced*) described a series of 40 cases of unexplained heart failure in African troops, mostly from West Africa, serving in the Middle East. Their ages were usually between 20 and 30, and all but two were under 40. They were admitted to hospital either with severe left heart failure or with combined pulmonary and systemic congestion and ascites. The history was of increasing dyspnœa for a few weeks or months, and sometimes of attacks of nocturnal dyspnœa with hæmoptysis. The main clinical

findings were normal rhythm with a very small rapid pulse, and often extrasystoles. Alternation was the rule. The heart was grossly enlarged to the left, and the sounds distant with a triple rhythm at the apex; systolic murmurs were common and the pulmonary second sound was much accentuated. The blood pressure was often low, below 120 mm. systolic, but sometimes slightly raised to 140 to 160 systolic. The diastolic was relatively high, often 100 mm. or just over. X-rays showed gross general enlargement of the heart with a prominent pulmonary artery and conus, and in half the cases the aorta was unduly small.

The Kahn was positive in about half, but this was common in African patients and ascribed to previous infection with yaws. The urine was usually normal and there was no evidence of nephritis. Blood examination rarely showed any anæmia and sickling was excluded, but eosinophilia was sometimes present. Ankylostomiasis and other parasitic infestations were common. There was no evidence of avitaminosis, no peripheral neuritis, and no malnutrition. The diet was adequate and far better than they were accustomed to at home.

There was no response whatever to thiamin given in adequate doses, both intravenously and by mouth. The response to digitalis and mersalyl was moderate. Some improved and became free from congestive heart failure, though they could do little; in these the heart diminished in size, though remaining considerably enlarged. Seventeen patients had died in hospital and necropsies were done in all these.

The hearts showed gross enlargement involving all cavities, and due more to dilatation than hypertrophy, though the weight was increased. There was no valvular disease, except in two cases with old mitral endocarditis and thickening. The coronary arteries were healthy in all. In three cases there was marked hypoplasia of the aorta, but the size of the aorta had not been specially noted in the earlier necropsies. In some cases there was an obvious and extensive subendocardial fibrosis, with fibrous areas resembling shallow infarcts in the ventricles, adherent to which was organized antemortem clots. Histological examination was still in progress, but some sections examined had shown extensive subendocardial necrosis and fibrosis without appreciable inflammatory reaction.

The ætiology was briefly discussed in relation to hypoplasia of the aorta, primary myocardial failure of French authors, "isolated myocarditis," hypertension, nutritional deficiencies, and tropical diseases. The clinical and pathological findings corresponded to cases described in America of recent years under the heading of "isolated myocarditis" or Fiedler's myocarditis, and especially to those described by Smith and Furth in 1943. The possibility of heart failure being the end result of previous nutritional deficiency had to be considered, as had a constitutional factor associated with aortic hypoplasia.

Thoraco-Lumbar Sympathectomy and Hypertension

W. T. Cooke and J. A. Barclay (*introduced*) presented the results of thoraco-lumbar sympathectomy (performed by W. H. Sweet) in 23 cases with hypertension. According to the criteria adopted by Keats, Wagener, and Barker, ten were Grade 4, six Grade 3, and the remaining seven Grade 2. Encouraging results were noted in Grades 3 and 4, twelve to forty months after operation. Two cases died before the completion of the second stage and one twelve months later. The remainder showed marked symptomatic improvement, resolution of eye changes, and a return to normal blood pressure in three. Studies of renal function in 5 patients, before and after operation, and 4 further patients before operation, showed a marked fall in Tm. The ratio of blood flow to Tm, however, was increased in varying degree in all cases except one in whom renal biopsy showed marked arteriosclerotic changes. Operation did not affect the ratio. The findings supported the observations of others, that the renal changes were probably not a primary factor in the genesis of essential and malignant hypertension.

Dextrocardia from Eventration of the Diaphragm

J. K. Rennie described a woman who had been suffering from bronchitis for three weeks. Dullness had been noted at the left base and was ascribed to fluid. She was a frail woman of 69 who had never suffered from any serious illness except she had a gastro-enterostomy performed for suspected duodenal ulcer 22 years before. The chest was very narrow, with

much kyphosis. On the right side in the fifth interspace a pulsation was apparent four inches from the midsternal line, which on palpation was found to be undoubtedly the apex beat. There was dullness half-way up the left back with absent respiratory murmur. The cardiogram proved to be normal. X-ray revealed a typical Petit's eventration of the left diaphragm with pronounced displacement of the heart to the right (plates shown revealing characteristic deformity). Displacement of the heart to the right is, of course, a constant finding in eventration of the left diaphragm but this is generally remarkably small in extent, even in middle-aged patients. This case shows how dextrocardia may be simulated, especially in the elderly with the chest changes described for this patient.

Acute Pericarditis in Still's Disease

Bernard Schlesinger mentioned that an adherent pericardium was not an uncommon finding at autopsy in the earliest accounts of this disease, but it was remarkable how this had rarely given rise to symptoms or signs during life, and for this reason it had never been suspected. He then described four cases that began with high fever, great distress, and evidence of acute pericarditis. This was subsequently followed by glandular enlargement, splenomegaly, a rash, and general rheumatoid involvement of the joints. A high leucocytosis was one of the features. Pericarditis thus appeared to be a definite and important part of the clinical picture of rheumatoid arthritis in childhood. The sequence of events favoured some specific infection, as yet unidentified, as the cause of rheumatoid arthritis. Finally he concluded that the separation of rheumatic fever and rheumatoid arthritis into two watertight compartments might not be justified and should receive further consideration.

INDEX OF AUTHORS

A

Aidaros, S. M. (and D. Evan Bedford and B. Girgis): Bilharzial heart disease in Egypt. 87.
Anrep, G. V. (and G. S. Barsoum, M. R. Kenawy, and G. Misrahy): *Ammi visnaga* in the treatment of the anginal syndrome. 171.
Ashworth, H. (and A. Morgan Jones): Aneurysmal dilatation of the left auricle with erosion of the spine. 207.

B

Barrett, A. M. (and Leslie Cole): Pulmonary vascular sclerosis with right ventricular failure. 76.
Barsoum, G. S. (and G. V. Anrep, M. R. Kenawy, and G. Misrahy): *Ammi visnaga* in the treatment of the anginal syndrome. 171.
Bedford, D. Evan (and S. M. Aidaros and B. Girgis): Bilharzial heart disease in Egypt. 87.
Berliner, Kurt: Auricular premature systoles: the duration of the electrical systole. 69.
Biörck, Gunnar: Hypoxæmia tests in coronary disease. 17.
Bourne, Geoffrey (and P. J. W. Mills): Dissecting aneurysm with cardiograms suggestive of cardiac infarction. 181.
Braun, K.: Paravertebral block and the electrocardiogram in angina pectoris. 47.
Brigden, Wallace (and Terence East): Postural hypotension. 103.
Burger, H. C. (and J. B. Van Milaan): Heart-vector and leads. 157.
Burwell, C. Sidney (and Richard H. Lyons): Induced changes in the circulation in constrictive pericarditis. 33.

C

Campbell, Maurice: Dissecting aneurysm with survival after rupture into the pleura. 200.
Cassidy, Maurice (and John Pinniger): Healed dissecting aneurysm. 130.
Cole, Leslie (and A. M. Barrett): Pulmonary vascular sclerosis with right ventricular failure. 76.

D

Daley, Raymond (and H. G. Miller): Angina pectoris with associated left paroxysmal ptosis. 29.
Decherd, George M. (and Arthur Ruskin): The mechanism of the Wenckebach type of A-V block. 6.
Drury, A. N. (and R. T. Grant and John Parkinson): Thomas Lewis (obituary). 1.
Duthie, R. J.: Mechanism of the Wolff-Parkinson-White syndrome. 96.

E

East, Terence (and Wallace Brigden): Postural hypotension. 103.
Ellis, Laurence B.: Electrocardiographic abnormalities in severe malnutrition. 53.
Evans, William: The heart in sternal depression. 162.

G

Gardner, Frances (and Jenner Hoskin): Silent dissection of the aorta. 141.
Girgis, B. (and D. Evan Bedford and S. M. Aidaros): Bilharzial heart disease in Egypt. 87.
Grant, R. T. (and A. N. Drury and John Parkinson): Thomas Lewis (obituary). 1.
Grossman, Nathan (and Lean Wallace): Præcordial electrocardiograms: a comparison of CF and V lead connections. 83.
Gutmann, David (and Robert Schnitzer): Myxœdema with pericardial effusion. 25.

H

Hartley, Ronald (and John Parkinson): Early diagnosis of rheumatic valvular disease in recruits. 212.
Herson, R. N. (and M. Symons): Ruptured congenital aneurysm of the posterior sinus of Valsalva. 125.
Hill, Athelstane: The genesis of the normal electrocardiogram. 147.
Hoskin, Jenner (and Frances Gardner): Silent dissection of the aorta. 141.

J

Jones, A. Morgan (and H. Ashworth): Aneurysmal dilatation of the left auricle with erosion of the spine. 207.
—— (and F. A. Langley): Chronic dissecting aneurysms. 191.

K

Kenawy, M. R. (and G. V. Anrep, G. S. Barsoum, and G. Misrahy): *Ammi visnaga* in the treatment of the anginal syndrome. 171.

L

Langendorf, R. (and S. S. Mintz): Premature systoles originating in the S-A node. 178.
Langley, F. A. (and A. Morgan Jones): Chronic dissecting aneurysms. 191.
Lyons, Richard H. (and C. Sidney Burwell): Induced changes in the circulation in constrictive pericarditis. 33.

M

Miller, H. G. (and Raymond Daley): Angina pectoris with associated left paroxysmal ptosis. 29.
Mills, P. J. W. (and Geoffrey Bourne): Dissecting aneurysm with cardiograms suggestive of cardiac infarction. 181.
Mintz, S. S. (and R. Langendorf): Premature systoles originating in the S-A node. 178.
Misrahy, G. (and G. V. Anrep, G. S. Barsoum, and M. R. Kenawy): *Ammi visnaga* in the treatment of the anginal syndrome. 171.
Moir, Raymond A. (and K. Shirley Smith): Cardiovascular diseases in the British Army overseas. 110.

N

Nissim, J. A.: Dissecting aneurysm of the aorta: a new sign. 203.

P

Parkinson, John (and A. N. Drury and R. T. Grant): Thomas Lewis (obituary). 1.
—— (and Ronald Hartley): Early diagnosis of rheumatic valvular disease in recruits. 212.
Pinniger, John (and Maurice Cassidy): Healed dissecting aneurysm. 130.

R

Ruskin, Arthur (and George M. Decherd): The mechanism of the Wenckebach type of A-V block. 6.

S

Schnitzer, Robert (and David Gutmann): Myxœdema with pericardial effusion. 25.
Smith, K. Shirley (and Raymond A. Moir): Cardiovascular diseases in the British Army overseas. 110.
Stokes, Wilfred: The effect of nitrite and exercise on the inverted T wave. 62.
Symons, M. (and R. N. Herson): Ruptured congenital aneurysm of the posterior sinus of Valsava. 125.
Szekely, P.: The action of magnesium on the heart. 115.

V

Van Milaan, J. B. (and H. C. Burger): Heart-vector and leads. 157.

W

Wallace, Lean (and Nathan Grossman): Præcordial electrocardiograms: a comparison of CF and V lead connections. 83.

INDEX OF SUBJECTS

A

Abnormalities, electrocardiographic, in severe malnutrition (L. B. Ellis). 53.
Action of magnesium on the heart (P. Szekely). 115.
Ammi visnaga in the treatment of the anginal syndrome (G. V. Anrep, G. S. Barsoum, M. R. Kenawy, and G. Misrahy). 171.
Aneurysmal dilatation of the left auricle with erosion of the spine (H. Ashworth and A. Morgan Jones). 207.
Aneurysm, dissecting (see dissecting aneurysm).
Aneurysm, ruptured congenital, of the posterior sinus of Valsalva (R. N. Herson and M. Symons). 125.
Anginal syndrome, *ammi visnaga* in the treatment of the (G. V. Anrep, G. S. Barsoum, M. R. Kenawy, and G. Misrahy). 171.
Angina pectoris with associated left paroxysmal ptosis (H. G. Miller and R. Daley). 29.
Angina pectoris, paravertebral block and the electrocardiogram in (K. Braun). 47.
Aorta, dissecting aneurysm of the: a new sign (J. A. Nissim). 203.
Aorta, silent dissection of (J. Hoskin and F. Gardner). 141.
Army, cardiovascular diseases in the British, overseas (R. A. Moir and K. Shirley Smith). 110.
Auricle, aneurysmal dilatation of the left, with erosion of the spine (H. Ashworth and A. Morgan Jones). 207.
Auricular premature systoles: the duration of the electrical systole (K. Berliner). 69.
A-V block, mechanism of the Wenckebach type of (G. M. Decherd and A. Ruskin). 6.

B

Bilharzial heart disease in Egypt: cor pulmonale due to bilharzial pulmonary endarteritis (D. Evan Bedford S. M. Aidaros, and B. Girgis). 87.
Block, paravertebral, and the electrocardiogram in angina pectoris (K. Braun). 47.
British Army overseas, cardiovascular diseases in the (R. A. Moir and K. Shirley Smith). 110.
British Cardiac Society, proceedings of, 233.

C

Cardiac infarction, dissecting aneurysm with cardiograms suggestive of (G. Bourne and P. J. W. Mills). 181.
Cardiac Society, proceedings of, 233.
Cardiograms suggestive of cardiac infarction, dissecting aneurysm with (G. Bourne and P. J. W. Mills). 181.
Cardiovascular diseases in the British Army overseas (R. A. Moir and K. Shirley Smith). 110.
CF and V lead connections, a comparison of (L. Wallace and N. Grossman). 83.
Chronic dissecting aneurysms (A. Morgan Jones and F. A. Langley). 191.
Circulation in constrictive pericarditis, induced changes in (R. H. Lyons and C. S. Burwell). 33.
Congenital aneurysm, ruptured, of the posterior sinus of Valsalva (R. N. Herson and M. Symons). 125.
Constrictive pericarditis, induced changes in circulation in (R. H. Lyons and C. S. Burwell). 33.
Coronary disease, hypoxæmia tests in (G. Biörck). 17.
Cor pulmonale due to bilharzial pulmonary endarteritis (D. Evan Bedford, S. M. Aidaros, and B. Girgis). 87.

D

Depression, the heart in sternal (W. Evans). 162.
Diagnosis, early, of rheumatic valvular disease in recruits (J. Parkinson and R. Hartley). 212
Dilatation, aneurysmal, of the left auricle with erosion of the spine (H. Ashworth and A. Morgan Jones). 207.
Dissecting aneurysm of the aorta: a new sign (J. A. Nissim). 203.
Dissecting aneurysm with cardiograms suggestive of cardiac infarction (G. Bourne and P. J. W. Mills). 181.
Dissecting aneurysm, healed (M. Cassidy and J. Pinniger). 130.
Dissecting aneurysm with survival after rupture into the pleura (M. Campbell). 200.
Dissecting aneurysms, chronic (A. Morgan Jones and F. A. Langley). 191.
Dissection of the aorta, silent (J. Hoskin and F. Gardner). 141.

E

Early diagnosis of rheumatic valvular disease in recruits (J. Parkinson and R. Hartley). 212.
Effect of nitrite and exercise on the inverted T wave (W. Stokes). 62.
Egypt, Bilharzial heart disease in: cor pulmonale due to bilharzial pulmonary endarteritis (D. Evan Bedford, S. M. Aidaros, and B. Girgis). 87.
Electrical systole, duration of (K. Berliner). 69.
Electrocardiogram, the genesis of the normal (A. Hill). 147.
Electrocardiogram, paravertebral block and the, in angina pectoris (K. Braun). 47.
Electrocardiograms, præcordial: a comparison of CF and V lead connections (L. Wallace and N. Grossman). 83.
Electrocardiographic abnormalities in severe malnutrition (L. B. Ellis). 53.
Endarteritis, cor pulmonale due to bilharzial pulmonary (D. Evan Bedford, S. M. Aidaros, and B. Girgis). 87.
Erosion of the spine, aneurysmal dilatation of the left auricle with (H. Ashworth and A. Morgan Jones). 207.
Exercise, effect of nitrite and, on the inverted T wave (W. Stokes). 62.

G

Genesis of the normal electrocardiogram (A. Hill). 147.

H

Healed dissecting aneurysm (M. Cassidy and J. Pinniger). 130.
Heart, action of magnesium on the (P. Szekely). 115.
Heart block, A-V, mechanism of the Wenckebach type of (G. M. Decherd and A. Ruskin). 6.
Heart disease, bilharzial, in Egypt: cor pulmonale due to bilharzial pulmonary endarteritis (D. Evan Bedford, S. M. Aidaros, and B. Girgis). 87.
Heart in sternal depression (W. Evans). 162.
Heart-vector and leads (H. C. Burger and J. B. Van Milaan). 157.
Hypotension, postural (T. East and W. Brigden). 103.
Hypoxæmia tests in coronary disease (G. Biörck). 17.

I

Induced changes in the circulation in constrictive pericarditis (R. H. Lyons and C. S. Burwell). 33.
Infarction, dissecting aneurysm with cardiograms suggestive of cardiac (G. Bourne and P. J. W. Mills). 181.
Inverted T wave, effect of nitrite and exercise on the (W. Stokes). 62.

L

Lead connections, a comparison of CF and V (L. Wallace and N. Grossman). 83.
Leads, heart-vector and (H. C. Burger and J. B. Van Milaan). 157.
Lewis, Thomas (obituary) (A. N. Drury, R. T. Grant, and J. Parkinson). 1.

M

Magnesium, action of, on the heart (P. Szekely). 115.
Malnutrition, electrocardiographic abnormalities in severe (L. B. Ellis). 53.
Mechanism of the Wenckebach type of A-V block (G. H. Decherd and A. Ruskin). 6.
Mechanism of the Wolff-Parkinson-White syndrome (R. J. Duthie). 96.
Myxœdema with pericardial effusion (R. Schnitzer and D. Gutmann). 25.

N

Nitrite and exercise, effect of on the inverted T wave (W. Stokes). 62.
Normal electrocardiogram, genesis of the (A. Hill). 147.

O

Overseas, cardiovascular diseases in the British Army (R. A. Moir and K. Shirley Smith). 110.

P

Paravertebral block and the electrocardiogram in angina pectoris (K. Braun). 47.
Paroxysmal ptosis, angina pectoris with associated (H. G. Miller and R. Daley). 29.
Pericardial effusion, myxœdema with (R. Schnitzer and D. Gutmann). 25.
Pericarditis, induced changes in the circulation in constrictive (R. H. Lyons and C. S. Burwell). 33.
Pleura, dissecting aneurysm with survival after rupture into the (M. Campbell). 200.
Postural hypotension (T. East and W. Brigden). 103.
Præcordial electrocardiograms: comparison of CF and V lead connections (L. Wallace and N. Grossman). 83.
Premature systoles, auricular: the duration of the electrical systole (K. Berliner). 69.
Premature systoles originating in the S-A node (R. Langendorf and S. S. Mintz). 178.
Ptosis, angina pectoris with associated paroxysmal (H. G. Miller and R. Daley). 29.
Pulmonary endarteritis, cor pulmonale due to bilharzial (D. Evan Bedford, S. M. Aidaros, and B. Girgis). 87.
Pulmonary vascular sclerosis with right ventricular failure (A. M. Barrett and L. Cole). 76.

R

Recruits, early diagnosis of rheumatic valvular disease in (J. Parkinson and R. Hartley). 212.
Rheumatic valvular disease in recruits, early diagnosis of (J. Parkinson and R. Hartley). 212.
Ruptured congenital aneurysm of the posterior sinus of Valsalva (R. N. Herson and M. Symons). 125.
Rupture into the pleura, dissecting aneurysm with survival after (M. Campbell). 200.

S

S-A node, premature systoles originating in the (R. Langendorf and S. S. Mintz). 178.
Sclerosis, pulmonary vascular, with right ventricular failure (A. M. Barrett and L. Cole). 76.
Silent dissection of the aorta (J. Hoskin and F. Gardner). 141.
Sinus, posterior, of Valsalva, ruptured congenital aneurysm of the (R. N. Herson and M. Symons). 125.
Spine, aneurysmal dilatation of the left auricle with erosion of the (H. Ashworth and A. Morgan Jones). 207.
Sternal depression, the heart in (W. Evans). 162.
Systole, duration of the electrical (K. Berliner). 69.

T

T wave, the effect of nitrite and exercise on the inverted (W. Stokes). 62.

V

Valsalva, ruptured congenital aneurysm of the posterior sinus of (R. N. Herson and M. Symons). 125.
Valvular disease in recruits, early diagnosis of rheumatic (J. Parkinson and R. Hartley). 211.
Vascular sclerosis, pulmonary, with right ventricular failure (A. M. Barrett and L. Cole). 76.
V and CF lead connections, a comparison of (L. Wallace and N. Grossman). 83.
Ventricular failure, right, pulmonary vascular sclerosis with (A. M. Barrett and L. Cole). 76.

W

Wenckebach type of A-V block, the mechanism of (G. M. Decherd and A. Ruskin). 6.
Wolff-Parkinson-White syndrome, mechanism of the (R. J. Duthie). 96.

PRINTED IN GREAT BRITAIN BY WILLIAM CLOWES AND SONS, LIMITED, LONDON AND BECCLES

SANDOZ PRODUCTS LTD.

Present

Two naturally occurring Cardio-active Glycosides

STROPHOSID

A new glycoside, k-strophanthoside, isolated from the seeds of Strophanthus kombé in a pure crystalline state and with its initial constitution unchanged.

It has a great advantage over other strophanthus glycosides, which are amorphous, in that it is obtained in a crystalline form, guaranteeing purity and constancy of action.

Strophosid is more rapid in action than digitalis, and while it is more toxic, it is less cumulative. Unlike digitalis, Strophosid is also active in cases of heart failure in which there is little or no compensatory hypertrophy of the heart.

Available in 1 c.c. ampoules containing 0·5 mg.
Boxes of 3, 6 and 50.

CEDILANID

Pure crystalline lanatosid C isolated from Digitalis lanata, and which is not contained in Digitalis purpurea.

Recent investigations have demonstrated that there is no fixed relationship between the toxicity of digitalis preparations and their therapeutically useful properties, so far as cardiac efficiency is concerned. Cedilanid, out of a large series of clinically useful cardiac glycosides, has been shown to possess the optimum ratio of toxic to therapeutic dosages.

Cedilanid increases cardiac efficiency in doses which do not influence the heart rate to any marked degree and therefore offers advantages in the treatment of heart failure where excessive slowing is not required. On the other hand, increased dosages produce the characteristic slowing of the heart rate typical of digitalis.

Supplied in two

Oral Solution (1 c.c. = 1 mg.).

Ampoules of ½ c.c. and 1 c.c. containing 0·4 and 0·8 mg. respectively.

FULL PARTICULARS FROM:—

Sandoz Products Ltd., 134 Wigmore St., London, W.1 SANDOZ

There is considerable evidence of the outstanding value of Cardophylin in producing vasodilatation. The coronary vasodilatation is manifested in an increased coronary blood flow and a beneficial effect on the myocardium; the renal vasodilatation is indicated by the powerful diuresis, while its antispasmodic action appears to be largely the result of the bronchodilatation induced by the drug.

Cardophylin

A SPECIALLY PREPARED COMPOUND OF THEOPHYLLINE-ETHYLENEDIAMINE

INDICATIONS

DISEASES OF THE CARDIOVASCULAR SYSTEM · OEDEMA · ASTHMA

LITERATURE AND SAMPLES ON REQUEST

In tablets (containing 0.1 gm.) for oral use. Suppositories (containing 0.36 gm.). Ampoules: Intramuscular (containing 0.48 gm. in 2 c.c. water). Intravenous (containing 0.24 gm. in 10 c.c. water).

MANUFACTURED BY

WHIFFEN & SONS LTD · CARNWATH ROAD

FULHAM · LONDON · S.W.6

PRINTED IN GREAT BRITAIN BY WILLIAM CLOWES AND SONS, LIMITED, LONDON AND BECCLES.